THE
LARYNGOSCOPE.

VOL. LXIX FEBRUARY, 1959 No. 2

MAXILLECTOMY IN CARCINOMA OF THE ANTRUM.*

An Analysis of 81 Cases and a Description of Certain Modifications of the Basic Technique of Extirpation.†

HAROLD G. TABB, M.D.,

New Orleans, La.

Many years ago a very brilliant surgeon, whose name regrettably I cannot recall, made a very wise observation to the effect that repetition of the same ideas does not necessarily mean paucity of ideas. It may quite as well, he said, mean intensity of conviction. On that basis, I return again to the subject of carcinoma of the antrum, to stress several of the things which I have already said in previous papers on this subject.

My thesis is briefly this:

1. At the best, the results in malignant disease of the antrum will never be very good, because the cards, to use the vernacular, are stacked against both patient and physician. The structural continuity of the area is an invitation to the rapid spread of disease processes. When the ethmoid area is involved, brain tissue is only a few millimeters away; furthermore, malignant growths in this area are predominantly of the epidermoid or squamous-cell variety and are usually of a high grade of malignancy.

---

*Read at the Meeting of the Eastern Section, American Laryngological, Rhinological and Otological Society, New York, N. Y., Jan. 8, 1959.

†From the Department of Otolaryngology, Tulane University of Louisiana School of Medicine.

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication Jan. 19, 1959.

2. Bleak as is this outlook, we should not accept the situation with an attitude of frustration and futility; instead, we should regard it as a challenge. The regional anatomy is immutably fixed, but immediate and long-term results can be improved if all physicians will employ a high index of diagnostic suspicion, and if all surgeons who encounter the disease will treat it with imagination and courage. Neither of these objectives is impossible of attainment.

DIAGNOSTIC CONSIDERATIONS.

Patients with carcinoma of the antrum are seldom seen early. A delay of three months after the onset of symptoms is average. A delay of six to nine months is frequent, and delays of a year or more are not infrequent. This is an insidious disease, and the same reasons that make patients delay in seeking medical consultation make physicians delay in diagnosing it.

The etiology of antral cancer is still unsettled, but there is no doubt that it is definitely related to, and often appears in association with, diseases of the nasal tract and paranasal sinuses which are benign in most cases, and which it is the tendency to regard as benign in all cases. The patients have become accustomed to the existence of these diseases. The physicians whom they consult are accustomed to managing them as benign, and they treat the patients, therefore, with the purpose of relieving symptoms without investigating the pathologic process. Otolaryngologists often fall into this error.

These are serious fallacies. Chronic sinusitis, various allergies, polyps and nasal obstructions all interfere with local ventilation and drainage and create what might be termed the climate of malignancy. Because of the frequency with which nasal and other polyps prove malignant on histologic examination, it is now our policy that all these and other masses, whether they are demonstrable grossly or can be demonstrated only by roentgenograms, should be removed and examined under the microscope. There is a dangerous impression among many otolaryngologists, as well as among other physicians, that a projecting mass in the sinuses, if it

is smoothly contoured and is not producing symptoms, is harmless and requires no treatment. This is not a safe policy. Similarly, we have come to believe that it is wise to remove all severe obstructive deviations of the nasal septum, because of the conditions which they set up by their interference with normal ventilation and function.

The wisdom of this policy was brought sharply home to me by the case of a 39-year-old white man who underwent routine submucous resection for a severe deformity of the septum. Under the obvious lesion lay an early carcinoma which was apparently just beginning to invade the maxillary sinuses. Radical maxillectomy was performed a few days after the first operation, and the patient, seven years and four months later, is still living and well. His case is one of the few that *does* prove, and one of the larger number that *could* prove, that carcinoma of the antrum is a curable disease when it is found early enough to permit radical removal of the growth with an adequate margin of normal tissue around it.

A far less happy instance was also an accidental diagnostic discovery. A 16-year-old Negro, hospitalized for a football injury of the orbital, nasal and ethmoid bones, had a large, mucosa-covered mass in the right nostril. It was regarded as a hematoma until biopsy prove it to be malignant. Radical maxillectomy was performed, but the growth was a rapidly growing rhabdomyosarcoma, and the boy died two months later.

Diagnostic Regimen.

Certain symptoms and signs require a diagnostic survey without delay, in spite of the fact that they are also, at least in their early, milder, manifestations, the symptoms and signs of malignant disease. They are as follows:

1. Unilateral nasal masses, with widening of the nasal bridge, or unilateral swelling and tenderness in the nasal area.

2. Polypoid masses in the nose and sinuses, particularly masses which have been present for a long time. Abnormally

free bleeding when they are removed should be viewed with deep suspicion.

3. Prolonged unilateral nasal obstruction, with a blood-tinged discharge or frank epistaxis.

4. An intractable purulent sinus infection which does not respond to adequate therapy.

5. Pain in the cheek or upper teeth out of proportion to the clinical findings.

6. Tender swellings of the palate and antral area.

7. Ocular proptoses.

As this list of symptoms and signs implies, the patient who suffers from them will not always be seen first by an oto-laryngologist. Dentists, ophthalmologists and other physicians must also come to realize the occasional lethal potentialities of these usually innocuous findings.

The steps of a correct diagnostic survey include:

1. A careful local examination.

2. Nasopharyngoscopy.

3. Roentgenologic examination. Roentgenograms in early malignant disease show only opacity, and the shadow cast by an antral cancer, which is a soft-tissue growth, cannot always be distinguished from the shadow cast by a benign lesion. Later, increased density of the sinuses or nasal cavities is followed by evidence of bony expansion or destruction.

4. Cytologic examination by the Papanicolaou technique. This is a misleading method unless it is constantly borne in mind that while a positive result is conclusive, a negative result is not, and must not be so regarded.

5. Aspiration biopsy, for which the same warning holds.

6. Surgical biopsy, repeated if it is negative.

7. Surgical exploration, which must be undertaken if the suspicion of cancer persists in the face of repeated negative biopsies. The pathologist must be supplied with a representa-

tive section of tissue and must be given ample time to study it. The surgery of antral cancer is radical mutilating surgery. It is disastrous not to employ it when malignant disease is present, but equally disastrous to employ it when it is not.

SURGICAL CONSIDERATIONS.

Over the last several years, there has come to be fairly general agreement that surgery, followed by irradiation, offers the best chance of survival for the patient with cancer of the antrum. The mortality of surgery was once distressingly high, immediately from shock and blood loss, and a little later from meningitis. Irradiation was, therefore, the method most generally used, as the lesser of two evils; but irradiation, without excision of the malignant growth, gives rise to serious complications, particularly when the bone has been invaded, and the surgical procedures necessary to control these complications must often be quite as radical as though surgery had been employed primarily.

Total removal of the maxillary sinus is necessary if cure is to be achieved in carcinoma of the antrum. Total removal of the sinus is impossible without total removal of the maxillary bone. Any surgery which is not based upon this fundamental concept is not adequate surgery. Surgery of such magnitude is now entirely practical, thanks to improved techniques of anesthesia, including hypotensive anesthesia; the liberal use of whole blood, now available in hospital blood banks and the development of efficient wide-spectrum antibiotics.

Basic Technique.

The basic technique of the radical operation for carcinoma of the antrum has already been published,[1] and I shall not describe it in detail. Instead, I shall merely list the principal steps of the operation and call attention to certain modifications employed in the most recently treated cases.

The Weber-Fergusson incision is used to expose the facial surface of the maxilla, and the entire maxillary bone, with the maxillary sinus, ethmoids, and orbital contents, is removed *en bloc*. To accomplish this, the palate is split in the midline, and the maxilla and portions of the zygoma and ethmoid bones

are separated from their attachments. The sphenoidal, frontal and posterior ethmoids are curetted and fulgurated, as are all other suspicious areas. It is generally preferable to include the eye in the dissection unless an undoubtedly clear margin of normal tissue can be demonstrated between it and the lesion. If there is any doubt, the eye must be sacrificed. During the operation, the entire cavity is irrigated with a cancerocidal solution, to reduce the number of viable cancer cells in the wound washings.

Modifications.

The following modifications of the original technique have been introduced over the last three years:

1. The electric saw instead of the Gigli saw is used to section the zygomatic bone, the infraorbital rim and the coronoid process of the mandible. With it, bone section is accomplished more rapidly and with less effort.

2. Ligation of the internal maxillary artery is still attempted routinely, but the omission of this step seems to be attended with little additional risk, and ligation is, therefore, abandoned if the artery lies deep to the pterygoid muscles and is difficult to identify.

3. We still prefer to employ the technique of hypotensive anesthesia devised by Adriani; but we have no illusions concerning its risks, and we now omit it in older patients or in patients who are poor surgical risks.

4. In the original technique, the mucosa over the hard palate was removed with the palate. We now salvage the membrane whenever the antral floor is not involved in the malignant process. It frequently is not, since most cancers in this anatomic area grow toward the nasal fossa or into the ethmoid area. This technique is practical only in edentulous patients, and it goes without saying that the cases must be selected very carefully. When it is used (see Fig. 1) the covering membrane is reflected toward the intact side and is retained *in situ* with the soft palate. The advantage of retention of the membrane is that it permits water-tight closure

of the surgical field and does away with the former necessity for a palatal prosthesis.

5. The cribriform plate was sometimes removed in the original technique. It is now removed with considerably greater frequency when it seems an inherent part of the lesion. This plate is the last bony barrier between the nasal sinuses and the cranial cavity; but when it is removed, the strong,

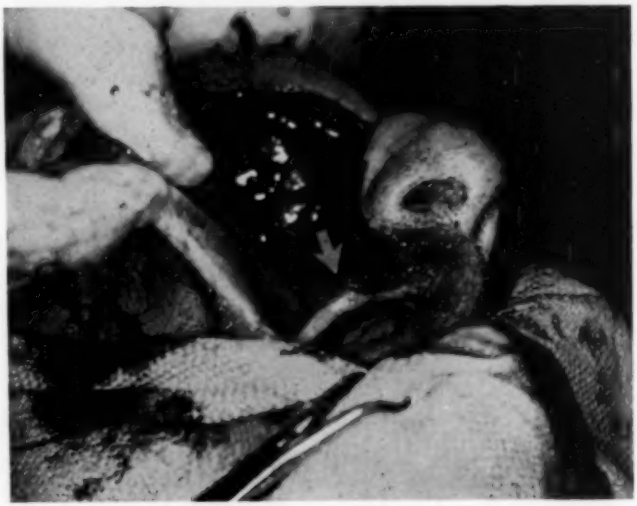

Fig. 1. Conservation of mucous membrane over bony palate in radical surgery for carcinoma of the antrum.

fibrous dura located between it and the brain tissue acts as a deterrent to the spread of the cancer. The plate is always opened when electrocoagulation of the posterior ethmoids is necessary, and when the orbital contents are removed with the maxillary bone. Curettement and electrocoagulation is employed when disease has extended to this area.

Whenever the dura is opened, accidentally or deliberately, there is leakage of cerebrospinal fluid, and the cranial cavity is thus laid open to the possible spread of infection from the

surgical field. The rent in the dura is closed by the application of gelatin foam overlaid by a skin graft; both materials are held in place with the medicated wool packing inserted at the end of the operation.

6. The mucous membrane over the septum adjacent to the surgical cavity is removed, even though there is no evidence of its involvement in the malignant process. Removal of

Fig. 2. Effective prosthesis for patient who has undergone radical surgery for carcinoma of the antrum (devised and constructed by Mr. John H. O'Donnell, prosthesist).

this membrane facilitates repacking during the postoperative period and makes it much less painful.

POSTOPERATIVE IRRADIATION.

Irradiation, applied by a competent radiotherapist in maximum doses, is pushed to the limit of tolerance after operation, in every case in which it is not absolutely certain that an entirely adequate margin of disease-free tissue has been removed with the excised specimen.

Dr. J. V. Schlosser, who treated practically all of the cases observed at Charity Hospital of Louisiana at New Orleans, has observed that oxygenation of the tissues to be treated just before they are irradiated appears to potentiate the effect of therapy by sensitizing the tumor cells and thus making the irradiation effects more lethal to them. The simultaneous use, in some cases, of oxygen and nitrogen mustard or other chemotherapeutic agents also seems to enhance the effects of irradiation. The perfusion techniques, which have given brilliant results in more anatomically favorable areas of the body, have still not been perfected clinically for use in the head and neck, but the outlook is promising.

These methods may eventually replace surgery, but their consummation lies in the future. For the present, surgery followed by irradiation is the method which lies at hand, and it should be used promptly and adequately. This is a mutilating method, but the mutilation can be quite effectively compensated for by well-made prostheses (see Figs. 2, 3), and it is the best method presently available for the salvage of life in a potentially highly lethal disease.

ANALYSIS OF CASES.

Over the 18-year period ending in December, 1958, I have been able to collect 81 cases of carcinoma of the antrum, 67 from Charity Hospital of Louisiana at New Orleans and the remainder from the Veterans' Administration Hospital and the Eye, Ear, Nose and Throat Hospital in New Orleans. Sixty of these cases, which have already been reported,[1] covered the 15-year period ending in June, 1955. The fact that 13 of the second series of 21 cases were observed in 1958 would probably not impress statisticians, but I take considerable encouragement from this recent concentration of cases, and from the fact that 15 of the 21 cases in the second series were referred cases.

Forty-three of the 67 patients from the New Orleans Charity Hospital were Negroes, which is in accord with the large Negro component in the hospital population. Twenty-nine of the 81 patients in the combined series were female, which is in accord with the generally reported sex incidence. The

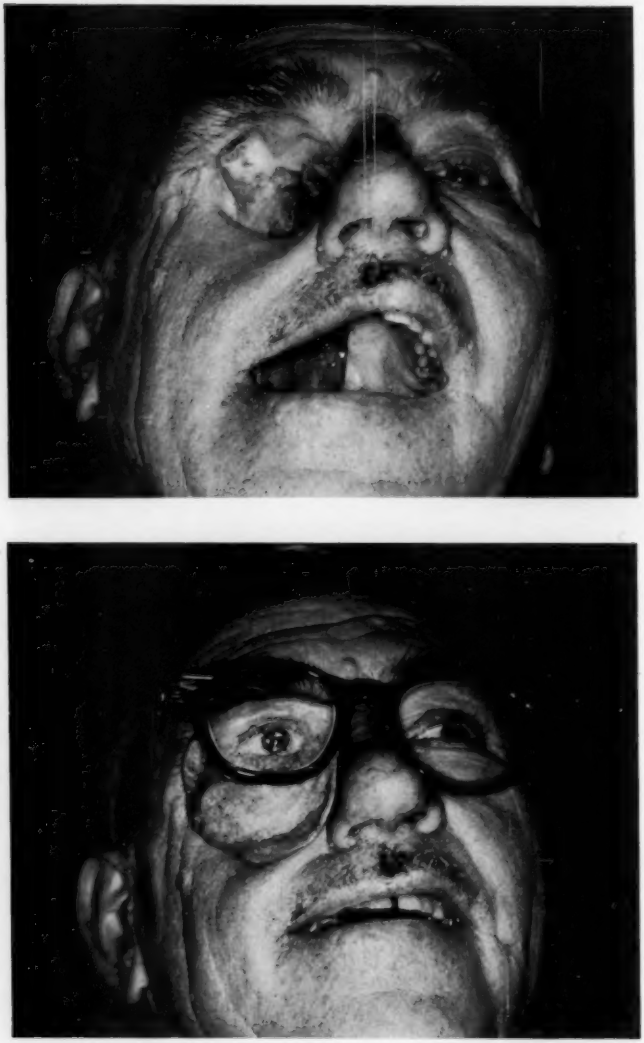

Fig. 3. Patient who has undergone radical surgery for carcinoma of the antrum.
A. Without prosthesis; B. Wearing prosthesis.

age range was from 16 to 77 years, and 58 patients were 50 years of age or older, which again is in accord with general observations.

In seven of the 81 cases in this series (see Fig. 4), the disease was so far advanced that no curative measures could be considered. All of the 27 patients treated by irradiation only were included in the first series of 60 cases reported in 1957,[1] except for one patient in the second series who would not

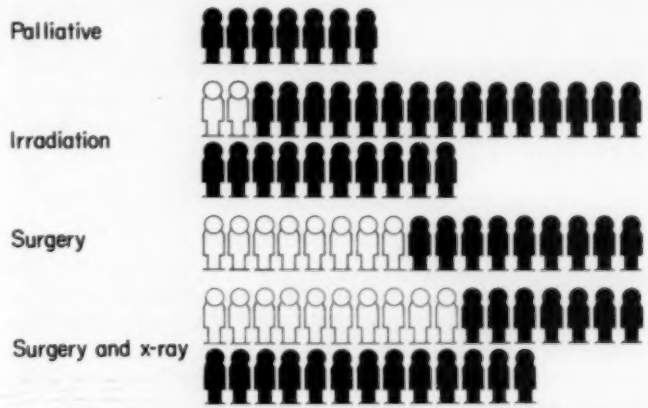

Fig. 4. Methods of treatment and mortality in 81 cases of carcinoma of the antrum, 1940-1958. Three of the patients still alive after combined surgery and X-ray have recurrent disease.

consider surgery. Seventeen patients were treated by surgery only, and 30, including most of the patients in the second series, by surgery followed by irradiation. In some instances the surgery was not adequate, because of errors of judgment on the part of the surgeon, or because the patients refused to permit the complete operation, or because the disease was too far advanced to accomplish all that was desirable.

All but 20 of these 81 patients are dead (see Fig. 4), and three of those still living have recurrent disease. Of the seven patients treated surgically in the second series who are still alive and well, only three were operated on more than

six months ago, and the longest period of survival is two-and-one-half years.

On the other hand, all seven of the patients in the first series who were reported to be alive and well in 1957 are still alive and well, one of them for almost 15 years, five between more than five and almost eight years, and the remaining patient for four years and nine months. One of these patients, who was operated on in March, 1953, had only a simple maxillectomy then and his eye was not removed. Recurrent disease was discovered when he reported for examination in August, 1957. Immediate radical maxillectomy was performed, followed by irradiation, and he is living and well 15 months after this second procedure.

Carcinoma of the antrum will probably never be anything but a depressing disease to treat, but, as these cases show, it is not entirely hopeless.

SUMMARY.

The recorded results of carcinoma of the antrum are discouraging, but they can be improved by a determined effort to make the diagnosis earlier and by a courageous surgical attack upon the disease process. Total removal of the sinus is impossible without total removal of the maxillary bone. Any surgery not based upon this fundamental concept is not adequate surgery. Unless it is entirely certain that the growth has been removed with a margin of disease-free tissue about it, the operation must be followed by irradiation pushed to the limits of tolerance.

Certain modifications of the surgical technique which have recently been introduced are described in this communication, and 21 addditional cases are added to the 60 cases reported in 1957.[1] None of the 10 surviving patients in the second series have been under observation long enough to permit any statement about end-results, but seven patients in the first series are alive and well for periods varying from four years and nine months to 14 years and nine months.

REFERENCE.

1. TABB, H. G.: Carcinoma of the Antrum. An Analysis of 60 Cases with Special Reference to Primary Surgical Extirpation. THE LARYNGOSCOPE, 57:269-341, April, 1957.

EARLY DIAGNOSIS AND TREATMENT OF CORROSIVE BURNS OF THE ESOPHAGUS.*

EUGENE L. YURICH, M.D.,

Detroit, Mich.

INTRODUCTION.

Early in 1957, the author reviewed cases of corrosive burns of the esophagus at this hospital and in the literature. In addition to the variable and controversial methods of therapy, other distressing facts were recognized. These included: *1.* Inadequate early diagnosis based mainly on presumptive evidence; *2.* Withholding early therapy because of an inadequate diagnosis; *3.* Institution of therapy without an accurate diagnosis; and *4.* Needlessly long hospital confinement in the majority of instances.

With the advent of Cortisone therapy and its acceptance in the treatment of esophageal burns, it became necessary to revamp our thinking on these matters, and we were prompted to institute earlier and more accurate means of diagnosis.

REVIEW OF THE LITERATURE.

Spain and his co-workers[1,2] in 1950, experimented with Cortisone as to its effects on granulation tissue formation in mice. In using high doses of Cortisone, they found that it unquestionably inhibits formation of granulation tissue. It was revealed, however, that Cortisone was effective only in the early stages of involvement and, if instituted later than 48 hours after the tissue injury the result was unsatisfactory. Rosenberg and his associates[3] in 1951, induced lye burns in the esophagi of rabbits, followed by immediate treatment (within 48 hours) with Cortisone. In their series, a marked decrease in fibroplasia occurred with a significantly diminished scar tissue formation. There was a concomitant in-

*From the Department of Otolaryngology, Wayne State University Medical School and Receiving Hospital, Detroit, Mich.

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication Nov. 1, 1958.

crease in secondary infections, mostly intra-thoracic, in the treated cases. They repeated their experiments in 1953,[4] administering penicillin in conjunction with the Cortisone. There resulted a marked reduction of suppurative infections accompanying the lye burns, although five of 44 rabbits developed spontaneous perforation of the esophagus in the Cortisone-penicillin treated group. Weisskopf[5] in 1952, experimented with dogs having induced lye burns of the esophagus, using a terramycin-Cortisone combination therapy. In none of the treated cases did a stricture develop, whereas all the controls developed stricture after 35 days. He particularly stressed treatment within 48 hours, and antibiotics in combination with the Cortisone.

There were questions that arose concerning the value of ACTH either alone or in combination with Cortisone. With the former hormone, there is a stimulated production of all the adrenal steroids, including desoxycorticosterone. This latter steroid has been reported by Taubenhaus[6] actually to increase fibroplasia.

When to do an esophagoscopy imposes a question. Some authors[7,8,9,10] feel that early esophagoscopy is dangerous because of the possibility of perforating an already weakened esophageal wall. Others[11,12,13] are of the opinion that esophagoscopy should be done within 24 to 48 hours of the time of the burn. Still others[14,15] prefer to esophagoscope the patient from the fourth to the seventh day following the ingestion of the caustic agent.

PATHOLOGIC CONSIDERATIONS.

The usual areas of involvement in the esophagus are the cricopharyngeus, the crossing of the left main bronchus and the cardia. The upper esophagus is probably the most frequently involved area, whereas the lower esophagus is usually the most seriously involved, the latter being due to reflex spasm at the cardia.[7,16] The extent of injury to the esophagus is dependent upon the agent, duration of contact, concentration and the amount ingested. The severity of the burn may be diviided into three groups: *1.* First degree, which involves

only the mucosal and submucosal layers. These heal without stricture formation and present little problem in therapy. *2.* Second degree, in which the necrosis extends to the muscular wall. In this stage there is initial hyperemia and edema with subsequent ulceration in a few days.[7,17] By the second week, there is granulation tissue formation, which ultimately converts to fibrous connective tissue and stricture formation. Stricture may develop in three weeks, usually develops within eight weeks; however, on rare occasions it takes several months or even years to present itself.[7,11] *3.* Third degree burns are those with involvement of all layers of the esophagus with associated periesophagitis and mediastinitis. Mortality is high in this group.

Corrosive agents differ as to their effect on the esophagus. Caustic alkalis (*e.g.*, NaOH), produce a liquifactive necrosis, whereas acids (*e.g.*, HCl) produce a coagulation necrosis. Pyloric stenosis is more commonly seen in acid burns, since the alkalis are neutralized by the acidic gastric contents; however, pyloric stenosis can and does occur in alkali burns.[10,17]

PRESENT STUDY.

In the past 18 months (March, 1957, to August, 1958), 37 patients entered the emergency room at Receiving Hospital of Wayne State University with symptoms and signs suggestive of the ingestion of a corrosive agent. Of these, there were 28 (77 per cent) in the age group 10 months to 5 years, and six (16 per cent) in the age group 5 to 10 years. The remaining three (14 years, 26 years and 58 years, respectively) were suicide attempts. The agents taken by mouth were NaOH (27 cases, 75 per cent), lysol, two cases; clinitest tablets, two cases; ammonia (one case); chlorox (one case), and four cases in which the agent was undetermined.

By using early diagnostic pharyngoscopy and esophagoscopy, we were able to narrow the cases of actual burn down to 10 (27 per cent of the suspected 37 patients). Of these ten actual esophageal burns, seven were by NaOH, one by ammonia, one by lysol, and one by an undetermined agent.

Age Distribution:

Age	Suspected Burns No.	Suspected Burns %	Actual Burns (27%) No.	Actual Burns (27%) %
10 months to 5 years	28	77	8	80
6 years to 10 years	6	16	--	--
Over 10 years	3	7	2	20
Total	37	100	10	100

Agents Used:

Agent	Suspected Burns No.	Suspected Burns %	Actual Burns No.	Actual Burns %
NaOH	27	75	7	70
Lysol	2	5	1	10
Clinitest	2	5	--	--
Ammonia	1	2	1	10
Chlorox	1	2	--	--
Undetermined	4	11	1	10
Total	37	100	10	100

DIAGNOSIS.

Initially, the diagnosis is presumptive with a history of the ingestion of some corrosive agent. Pain about the mouth, tongue, throat and substernal areas, dysphagia and hoarseness, are symptomatic indications. The finding of burns about the lips, tongue, oropharynx and hypopharynx furnishes additional presumptive evidence of esophageal burns. These findings, however, do not confirm the presence of burns in the esophagus. Contrariwise, an absence of these findings does not obviate the possibility of burns. There are certain cases where the patient arrives in shock with extensive burns of the mouth and pharynx, and/or dyspnea from severe laryngeal burns. In these uncommon cases, the diagnosis becomes quite obvious. It is our belief that early roentgenograms and/or fluoroscopy are not valid either to prove or disprove an esophageal burn. Esophagoscopy is done usually within the first 24 hours. It is always done under general anesthesia; and once the burned area is visualized, the esophagoscope is withdrawn to preclude the possibility of perforating through the burned area.

In the untreated cases of esophageal burn, the patient usual-

ly recovers from his initial symptoms after the first week. He then passes into a latent period of from two to four weeks, when he has few or no symptoms. There then develops progressive dysphagia, first for solids and later for semi-solid foods. In uncommon instances, however, the progression may be so insidious as not to give symptoms for months or even years. Often a foreign body lodged in the esophagus may be the first indication of an old stricture. The diagnosis of chronic stricture is made by esophagoscopy and roentgenography, the latter being invaluable in determining the degree, area, tortuosity and length of the stricture.

TREATMENT.

A. Early Treatment:

Initial treatment is to neutralize the offending agent. For alkalis, acetic acid (1 to 2 per cent), and citrous juices are given in small repeated doses. For acids, olive oil, milk and egg whites are of value. Sodium bicarbonate is not used because of the gas produced with distension and possible induction of vomiting. Gastric tubes are not inserted either for lavage or for feeding purposes, as they add to the original trauma and may tend to incite vomiting with additional corrosive effects to the wall of the esophagus. Since the majority of the caustic agents are neutralized by the gastric contents, it seems illogical to lavage these patients. In the case of acid burns, the argument is not so strong against lavage. All in all, there is much controversy regarding lavage and gastric tubes.[7,10,18] Diet is progressed slowly beginning with clear liquids, then pureed foods, and by the end of the week, the patient can ordinarily tolerate semi-solids.

Measures for shock are instituted promptly when indicated, and tracheotomy is carried out if the situation requires it. Antibiotics are given because of the possibility of such complications as periesophagitis, mediastinitis, pulmonary infections, abscess formation and esophageal perforations. Other therapeutic measures include sedatives and analgesics in appropriate dosages.

Steroids are given in relatively large doses, preferably

within 24 hours, and always within 48 hours after ingestion of the corrosive agent. The average dose for the child in the 1 to 4 year group is 40 to 50 mgm. of prednisolone daily in four divided doses. The duration of therapy is dependent upon the response to medication as determined by repeat esophagoscopic examinations done at ten day intervals. The average duration of therapy in our series was two to three weeks.

Eight out of our ten cases of esophageal burns which were treated with steroids responded favorably. A typical case is presented:

E. E., a 3-year-old colored male with a history of ingesting "Drano" (NaOH). He arrived in the emergency room in only mild distress from his oral burns. Initial examination on July 15, 1957, revealed burns of the lips, tongue, and buccal mucosa. Esophagoscopy was done within 12 hours of admission and revealed an area of burn in the mid-esophagus at about the area of the crossing of the left bronchus. He was placed on penicillin and prednisolone (10 mgm. every six hours). Repeat esophagoscopy on July 26, 1957 (11 days after admission) revealed residual eschar in the mid-esophagus. Steroids were discontinued when a follow-up esophagoscopy on August 11, 1957, gave no evidence of residual burn. Two esophograms subsequently taken have revealed no tendency toward stricture formation.

Of our ten cases of esophageal burns, two developed strictures in spite of what was considered an adequate dose of steroids. The first was a short annular stricture of the mid-esophagus which responded well to peroral bougienage. The second case is presented:

O. A., a 2-year-old colored male, found by his mother on May 31, 1958, with a can of lye in his hands. He was crying, salivating and vomiting. On admission to the hospital, there were evident burns of the lips, tongue, buccal mucosa and oropharynx. He was moderately dyspneic on admission, but this proceeded to severe dyspnea within 12 hours, and a tracheotomy was performed. He was placed on intravenous feedings, analgesics and antibiotics. Esophagoscopy within 20 hours of injury revealed severe burns involving the hypopharynx, larynx and cervical esophagus. Hydrocortisone (50 mgm. every six hours) was instituted. On June 11, 1958, (ten days after admission) a repeat esophagoscopy revealed extensive eschar involving the cervical and lower mid-esophagus. On June 13, 1958, he developed a hemorrhagic gastritis. Upper GI series revealed a possible gastric ulcer and Cortisone was discontinued. On June 16, 1958, repeat upper GI X-rays were read as negative and Cortisone was reinstituted. On June 23, 1958, esophagoscopy again revealed persistent eschar in the cervical esophagus and lower mid-esophagus. On July 8, 1958, there was esophagoscopic evidence of stricture at the lower mid-esophagus. This was confirmed by fluoroscopy. Cortisone was discontinued. Gastrostomy was performed on July 18, 1958, and after waiting two weeks for healing of the wound, retrograde dilatations were insti-

tuted using Tucker bougies. Up to the present time, he is on such therapy.

In cases that are not diagnosed within the 48-hours "grace period," thereby obviating the use of Cortisone, the Salzer technique[19,20] is instituted. This consists of daily dilatations with Hurst mercury weighted bougies for three to four weeks and then at gradually decreasing intervals. The patient may be forced to continue this treatment for several years, even though feeling well. This forces the physician to be very persistent and encouraging.

B. Late Treatment:

1. Blind bougienage—This is mentioned only to be condemned, except in those cases where mercury weighted bougies are used to maintain patency of the lumen.

2. Peroral esophagoscopic bougienage[21]—This is excellent for short, single, annular strictures, but not of use in patients with multiple, long or tortuous strictures.

3. Peroral bougienage over a string[22]—The patient swallows a silk thread and, when anchored securely in the gut, a fenestrated olive-tipped bougie is passed over the thread using the latter as a guide.

4. Peroral bougienage over piano wire—This has been used very successfully in our clinic, using the Eder-Puestow dilator set. This consists of a piano wire with a flexible tip which can be passed easily through the esophagus. The wire acts as a guide for the staff and olive-tipped bougies which are passed over it. The advantage of this over string is that the passage of the wire and the dilatation may be carried out at the same clinic visit.

5. Retrograde bougienage[23]—This technique necessitates a gastrostomy, but it is very effective and is the least dangerous method of dilatation. It is especially useful in children and uncooperative adults. Treatment is long and tedious and may be followed by mercury weighted bougie dilatation.

6. Surgical repair of the esophagus—The two methods mainly used are esophagogastrostomy and esophagojejunostomy. It is generally felt, that if at all possible, dilatation is

preferential to surgery. There are some, however, who value surgery very highly in certain cases.[13,16,24]

COMMENT.

It is quite evident that a great deal of controversy remains regarding the diagnosis and treatment of esophageal burns. In stressing the use of early esophagoscopy, in no instance were there any untoward effects in our series. Since ulceration and sloughing do not usually take place until "after a few days,"[7,17] it appears logical to assume that the esophagus would resist more trauma initially than it would, for example, from the fourth to the seventh day. In instituting treatment on a presumptive basis alone, there is imposed upon the patient unnecessary hospital stay, financial obligations and medical therapy.

The average duration of stay for the suspected burn cases with negative esophagoscopic examinations was two or three days as compared to two or three weeks for the treated cases. In addition, it presents a needless worry to the patient and family regarding the possibility of stricture later in life.

The use of Cortisone is a rather recent adjunct in the treatment of esophageal burns, but it is rapidly becoming a more widely acceptable early form of therapy. It is not, however, the entire answer to the problem, as is evidenced by the two failures in our series even with apparently adequate dosages. There is the possibility, however, that the Cortisone did decrease in degree the amount of stricture, but this would be difficult to prove. We are convinced that Cortisone and its allied steroid compounds are of definite value in our armamentarium against the formation of esophageal strictures.

Our ultimate goal should be in the realm of prophylaxis by the prevention of such accidents through adequate education and enforced legislation. Until such time arrives, we shall be forced to search for new and better methods of therapy.

SUMMARY.

1. Treatment of corrosive burns of the esophagus is controversial.

2. Early esophagoscopy within 24 to 48 hours is stressed as a means of adequate and accurate early diagnosis.

3. The theories and experimental data supporting Cortisone therapy are presented.

4. Pathologic considerations are outlined.

5. Early and late treatment of esophageal burns are outlined with particular reference to Cortisone therapy.

6. A series of 37 suspected cases are reported, of which ten had actual burns by esophagoscopic examination. The burned cases were placed on Cortisone therapy. Eight patients responded favorably, whereas two developed strictures in spite of treatment.

7. A successful case with Cortisone therapy is presented.

8. A failure in Cortisone therapy is presented.

ACKNOWLEDGMENTS.

My sincere appreciation to Dr. James E. Croushore and other members of the staff for their cooperation in the preparation of this paper.

BIBLIOGRAPHY.

1. Spain, D.; Molomut, N., and Haber, A.: Biological Studies on Cortisone in Mice. *Science*, 112:335, 1950.

2. Spain, D.; Molomut, N., and Haber, A.: The Effect of Cortisone on the Formation of Granulation Tissue in Mice. *Amer. Jour. Pathol.*, 26:710, 1950.

3. Rosenberg, N.; Kunderman, P.; Vroman, L., and Moolten, S.: Prevention of Experimental Lye Strictures of the Esophagus by Cortisone. *Arch. Surg.*, 63:147, Aug., 1951.

4. Rosenberg, N.; Kunderman, P.; Vroman, L., and Moolten, S.: Prevention of Experimental Esophageal Stricture by Cortisone. *Arch. Surg.*, 66:593, 1953.

5. Weisskopf, A.: Effects of Cortisone on Experimental Lye Burn of the Esophagus. *Ann. Otol., Rhinol. and Laryngol.*, 61:681, Sept., 1952.

6. Taubenhaus, M., and Ambomin, G.: Influence of Steroid Hormones on Granulation Tissue. *Endocrinol.*, 44:359, April, 1949.

7. Stothers, H.: Chemical Burns and Strictures of the Esophagus. *Arch. Otolaryngol.*, 56:262, Sept., 1952.

8. Hanckel, R.: Some Observations Concerning the Salzer Method of Treatment of Lye Burns of the Esophagus. *South. Med. Jour.*, 39:263, March, 1946.

9. Crowe, J.: Poisoning Due to Lye; Value of Bokay Prophylactic Dilatation in Prevention of Early Strictures of the Esophagus. *Amer. Jour. Dis. Child.*, 68:9, July, 1944.

10. Hollinger, P., and Johnston, K.: Caustic Strictures of the Esophagus. *Ill. Med. Jour.*, 98:246, Oct., 1950.

11. Smith, V.; Compton, J., and Palmer, E.: Cortisone and Acute Lye Corrosion of the Esophagus. *Arch. Otolaryngol.*, 58:235, Sept., 1953.

12. Huizinga, E.: Observations on Caustic Burns of the Esophagus. *Acta Oto-Laryngol.*, (Supp.), 116:145, 1954.

13. Daly, J., and Cardona, J.: Corrosive Esophagitis. *Amer. Jour. Surg.*, 93:242, Feb., 1957.

14. McKibben, B., and Lee, S.: Stenosis of the Esophagus and Stomach Following the Ingestion of Corrosive Substances. *Arch. Otolaryngol.*, 61:2, Jan., 1955.

15. Davis, M.: Early and Late Management of Caustic Burns of the Esophagus. *Amer. Surg.*, 22:308, March, 1956.

16. Fatti, L.; Marchand, P., and Crawshaw, G.: The Treatment of Caustic Strictures of the Esophagus. *Surg., Gynecol. and Obstet.*, 102:195, Feb., 1956.

17. Morse, H.: Treatment of Acute Caustic Burns of the Esophagus. *Jour. Tenn. St. Med. Assoc.*, 49:297, Sept., 1956.

18. Hoag, C.: Benign Strictures of the Esophagus; New Method of Gradual Dilatation. *Ann. Otol., Rhinol. and Laryngol.*, 46:327, June, 1937.

19. Salzer, H.: Early Treatment of Corrosive Esophagitis. *Wien Med. Wchnschr.*, 33:307, April, 1920.

20. Salzer, H.: Early Bougienage of the Esophagus in Cases of Cauterization with Lye. *Wien Med. Wchnschr.*, 84:736, June, 1934.

21. Jackson, C., and Jackson, C. L.: "Bronchoscopy, Esophagoscopy and Gastroscopy." W. B. Saunders Co., Phila., 1934.

22. Vinson, P.: The Swallowed Silk Thread as a Guide in Esophageal Instrumentation. *Arch. Otolaryngol.*, 13:94, Jan., 1931.

23. Tucker, G.: Cicatricial Stenosis of the Esophagus with Particular Reference to Treatment by Continuous String Retrograde Bougienage with the Author's Bougie. *Ann. Otol., Rhinol. and Laryngol.*, 33:1180, Dec., 1924.

24. Pinkham, R.: Enteric Reconstruction of the Esophagus in Children. *Amer. Jour. Surg.*, 94:350, Aug., 1957.

9345 Rutherford Avenue.

THE FENESTRATION OPERATION: A PERSPECTIVE IN TIME.*†

ROBERT C. BILGER, Ph.D.,
ALLAN C. GOODMAN, Ph.D.,
and
THEO. E. WALSH, M.D.,
St. Louis, Mo.

This report is a continuation of a long-term study[1-9] (begun in January, 1945) of the results of tests of hearing conducted on all of the patients upon whom the fenestration operation has been performed by one of us (T.E.W.) at McMillan Hospital (Department of Otolaryngology, Washington University School of Medicine). It deals with the first 818 patients upon whom the fenestration operation was performed. We here compare with respect to hearing for spondaic words measured three months post-operatively, a large number of heretofore unreported cases, with the cases that have been described in the earlier reports. To make these comparisons, the 332 cases dealt with in the earlier reports are treated as a single group (Group I), and the 486 new cases are divided into two groups of similar size representing approximately equal intervals of time (Groups II and III).

In addition to the comparisons among Groups I, II, and III, the earlier data, from Group I, will be used to evaluate long-term effects with respect to the stability of *1.* operative results, in terms of hearing level for spondaic words; *2.* hearing level for spondaic words in unoperated, otosclerotic ears; and *3.* speech discrimination scores for both fenestrated and unoperated ears of otosclerotic patients.

*From Central Institute for the Deaf and Hofheimer Audiology Laboratory of the Department of Otolaryngology of Washington University. This research was partially supported by a grant (B-243) to Central Institute for the Deaf from the National Institute of Neurological Diseases and Blindness.

†The authors are indebted to Mrs. Nancie Siedler for careful tabulation of the data, and for preliminary calculations in preparation for the final statistical tests.

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication July 20, 1958.

The measure of hearing used is the threshold for spondaic words as determined with recorded speech materials.[10] The discrimination scores were obtained with the Rush Hughes recordings of PB Auditory Tests 9, 10, 11 and 12.[11]

Final hearing level‡ is used because in this context it is a more logical measure of the outcome of the fenestration operation than gain.[6] A discussion of this point is included in Appendix A.

RESULTS AND DISCUSSION.

COMPARISON AMONG GROUPS.

The purpose of the comparison among the three chronological groups is to determine whether there has been a significant improvement from group to group in mean hearing level measured three months after the fenestration operation and, if so, to describe this improvement.

TABLE I. DESCRIPTION OF GROUPS I, II, AND III.

	Group I	Group II	Group III
Dates	Jan., 1945, to July, 1949	Sept., 1949, to May, 1953	May, 1953 to July, 1956
Case Numbers	1-332	333-567	568-818
Sample Size	199	232	227
Mean Age	33.6	34.2	35.0
Variability in Age (S.D.*)	9.41	8.76	9.78
Age Range	7-64	13-64	15-61

*S.D. = Standard Deviation.

The three groups are described in Table I. Some cases were dropped from the analysis for the following reasons: Group I, 132 cases in which a cartilagenous stopple was used, and one case in which speech audiometry was not used; Group II, three cases for which there were no post-operative tests of hearing; and Group III, 14 cases in which a stapes mobilization procedure preceded the fenestration operation, nine cases in which there have been no post-operative tests

‡Final Hearing Level is the exact equivalent of Residual Hearing Loss used by Davis and Walsh.[6] The new term follows the recent recommendation of Davis, Parrack and Hoople[12] to substitute "hearing level" for "hearing loss" when referring to audiometric measurements of the status of hearing.

of hearing, and one case in which speech audiometry was not used.

The results of the comparison among groups with respect to mean hearing level measured three months after the fenestration operation are presented graphically in Fig. 1. In this figure, the time scale (abscissa) is in years, and the mean hearing level for each group is plotted at the midpoint

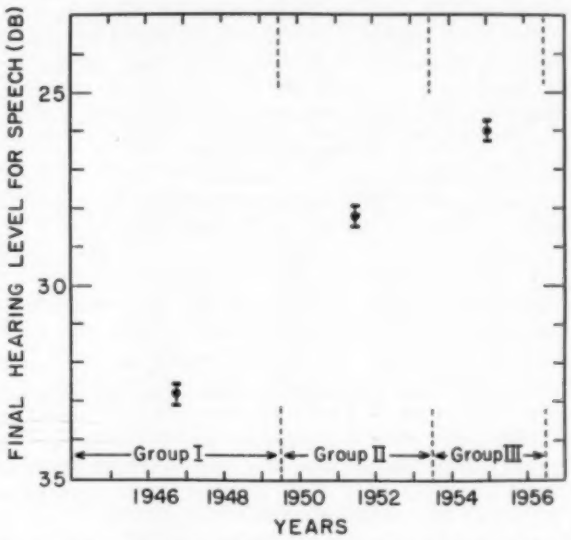

Fig. 1. Adjusted mean final hearing level for speech (spondaic words) as a function of chronological group (I, II, and III).

of the time interval during which the fenestration operations within each group were performed.

Figure 1 shows that the mean hearing level measured three months after the fenestration operation has improved by 3.5 db from Group I to Group II (from 32.7 to 29.2 db), and by 3.1 db from Group II to Group III (from 29.2 to 26.1 db). These changes are statistically significant beyond the 1 per cent level of confidence. This improvement is probably the

result of refinements in both surgical and diagnostic techniques.

Two features of the means presented in Fig. 1 are important; first, these means have been adjusted by the techniques of analysis of covariance to take into account the statistically significant difference between the groups with respect to preoperative hearing levels (Appendix A, Table A-1). This procedure is described in Appendix A. Second, *no cases were excluded from this analysis on the basis of the outcome of the*

TABLE II.

Percentage and Cumulative Percentages of Each Group Having the Indicated Post-Operative (3 Months) Hearing Level for Spondaic Words.

Hearing Level (db.)	Group I		Group II		Group III	
	Per cent	Cumulative per cent	Per cent	Cumulative per cent	Per cent	Cumulative per cent
20 db or better	3.0		12.1		28.2	
21-25	24.6	27.6	29.3	41.4	32.6	60.8
26-30	30.7	58.3	29.3	70.7	18.1	78.9
31-35	14.1	72.4	12.5	83.2	11.9	90.8
36-40	10.0	82.4	6.5	89.7	4.0	94.8
40 db or worse	10.6	93.0	7.3	97.0	4.0	98.8
No gain	7.0		3.0		1.2	

fenestration operation, so that the improvement from group to group of approximately 3 db represents the average of *all* cases, including the failures.

In order to determine where the improvement from group to group, with respect to hearing level measured three months after the fenestration operation, has been most pronounced, the percentage of each group attaining final hearing levels of 20 db or better, 21-35, 26-30, 31-35, 36-40, and 41 db or worse, as well as those in which there was no gain, was tabulated. The results of these tabulations are presented in Table II. In addition to the percentage of cases in each group with the specified hearing level, the percentage of each group with hearing level equal to or better than the indicated hearing level is presented in the column headed "cumulative per cent."

Davis and Walsh[6] report that 84.7 per cent of their cases (from Group I) had hearing levels better than 41 db three months after the fenestration operation. For the 199 cases called Group I in this report, 82.4 per cent had levels better than 41 db (see cumulative per cent for the 36-40 db interval of Group I in Table II). This slight discrepancy arises because the first five cases (No. 1 to No. 5), included in Group I, were not included in the sample of cases reported by Davis and Walsh.

Comparing the three groups with respect to the cumulative percentage of each group having final hearing levels better than 41 db (Table II), we see that the percentage of cases reaching this final hearing level increases from 82.4 per cent of Group I to 89.7 per cent of Group II to 94.8 per cent of Group III. When we compare the cumulative percentages of each group having final hearing levels better than 35, 30 and 25 db, the improvement from group to group is even more striking.

A comparison of the groups with respect to the percentage of cases that reached the theoretical "limit of improvement,"[6] a final hearing level of 20 db or better, shows that while only 3 per cent of Group I had final hearing levels of this magnitude, 12.1 per cent of Group II and 28.2 per cent of Group III received the maximum benefit from the fenestration operation.

At the other end of the hearing level continuum, there is a large decrease in the percentage of cases having final hearing levels worse than 35 db: 27.6 per cent of Group I compared to 16.8 per cent of Group II and only 9.2 per cent of Group III. The percentage of cases showing no gain as the result of the fenestration operation decreased from 7.0 per cent of Group I to 3.0 per cent of Group II and 1.2 per cent of Group III.

The hearing level measured three months after the fenestration operation is the only variable dealt with here. Even so, on the basis of the distributions of these hearing levels, it seems reasonable to attribute the increased percentage of cases having hearing levels better than 25 db to refinements

in surgical techniques, and the decreased percentage of cases having final hearing levels worse than 35 db to refinements in selection of cases and to better surgery also.

CLOSURES.

In an earlier report in this series, Davis, Walsh, and Eldert[7] observed that closures occurred in 22 of the 151 cases (14.6 per cent) from Group I in which there had been at least 10 db of improvement in hearing as the result of the fenestration operation. They suspected closure if the patient lost 10 db or more of hearing between the first (three months) and a subsequent post-operative test of hearing. They considered the closure to be confirmed when the patient gave a negative response to the fistula test. In every one of these cases, closure was confirmed by the end of the first year after the fenestration operation. The median post-operative interval at which closure was confirmed was 7.5 months, and the range of time over which closures were confirmed was from one to 12 months. The time-course for closure that they describe is consistent with their assumption that closure is related to the healing process.

As part of the present study, we tabulated the number of closures in Groups II and III, using the criteria listed by Davis, Walsh, and Eldert.[7] There were six closures among the 211 cases (2.8 per cent) in Group II in which there had been at least 10 db improvement in hearing. In Group III there were seven closures among the 215 cases (3.3 per cent) in which there had been 10 db of improvement. The six closures in Group II were confirmed at the following post-operative intervals: 6, 8, 9, 9, 10 and 11 months. For Group III the seven closures were confirmed at 6, 6, 6, 7, 7, 12 and 18(?) months post-operatively. In the case of the closure confirmed at 18 months, it is impossible to specify the time at which closure was complete, because the patient did not return for a check-up between three and 18 months after the operation.

When we compare the three groups with respect to the incidence of closure, we see that it drops sharply from 14.6 per cent in Group I to about 3 per cent in Groups II and III.

This decrease in the incidence of closures is almost certainly the result of modifications in surgical procedure.

The number of closures in Groups II and III, six and seven respectively, is too small to permit statistical comparisons of the time-course for closure; but since testing at six months was introduced into the follow-up routine, it should be mentioned that only four of the 13 closures in Groups II and III were confirmed by the end of six months after surgery. The timing of the second post-operative test between 7.5 and nine months after surgery might maximize the detection of closures at the second post-operative interval.

STABILITY OF HEARING IN FENESTRATED EARS.

Davis, Walsh, and Eldert[7] report that among successfully fenestrated ears (129 cases from Group I) a mean loss (decrement) in hearing, a threshold shift, of 2.8 db occurs during the first year after the operation. They attribute this rise in threshold to the normal healing process. It should be noted that the threshold shift and the closure both have been identified with normal healing, and both take place during the first year after surgery.

The purpose of this portion of the study was to determine whether hearing level in successfully fenestrated ears remains stable beyond the first year after the operation.

Stability of Hearing in Group I.

We used the data for the 43 patients in Group I whose hearing level three months after the operation was 35 db or better and who had returned for follow-up tests at the following post-operative intervals: three months, one year, three years, and seven years. The mean age for this sample from Group I, Subgroup I-A, at the time of the operation was 34.4 years.

The mean hearing level at each post-operative interval is plotted in Fig. 2 for Subgroup I-A. Statistical analysis (see Appendix B, Table B-1) of these data indicate that the loss of hearing for spondaic words that occurs during the first year is statistically significant beyond the 1 per cent level of confidence. The mean hearing levels at three and seven

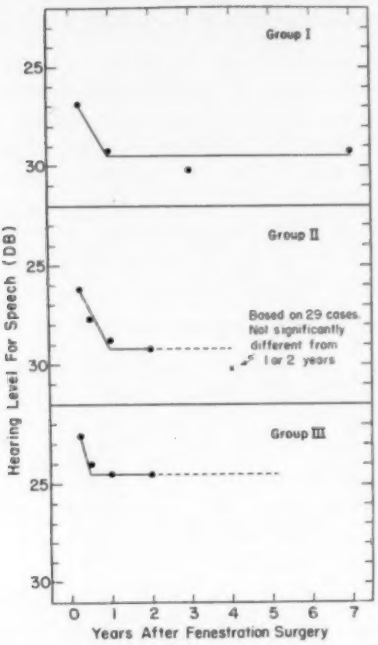

Fig. 2. Mean hearing level for speech (spondaic words) as a function of time after surgery for 43 successfully fenestrated ears from Group I (Subgroup I-A). The line represents an interpretation of the statistical analysis and is not fitted to the data.

Fig. 3. Mean hearing level for speech (spondaic words) as a function of time after surgery for 63 successfully fenestrated ears from Group II (Subgroup II-A). The point at four years after surgery represents 29 cases from Subgroup II-A (Subgroup II-B). The line represents an interpretation of the statistical analysis and is not fitted to the data.

Fig. 4. Mean hearing level for speech (spondaic words) as a function of time after surgery for 42 successfully fenestrated ears from Group III (Subgroup III-A). The line represents an interpretation of the statistical analysis and is not fitted to the data.

years, however, are not significantly different from the mean hearing level measured at the end of the first year after the operation. The solid line in Fig. 2 has been drawn to show that hearing got worse during the first year after the operation but remained constant thereafter. The threshold shift observed during the first year for Subgroup I-A of 2.5 db is only slightly smaller than that reported by Davis,

Walsh, and Eldert[7] for a different and larger sample from Group I.

It is possible that the procedure by which the 43 cases in Subgroup I-A were selected has produced a spurious result. To make sure that the result is correct, we retested the hypothesis that *hearing in successfully fenestrated ears remains stable beyond the first year after the operation.* To accomplish this retest, a new sample was selected from Group I, Subgroup I-B, that included all patients (113) whose hearing level three months after the operation was 35 db or better, and who had appeared for follow-up tests of hearing two years or more after the operation, as well as at three months and one year. The median post-operative interval at which the last test of hearing was obtained was 6.94 years. Closures were excluded from Subgroup I-B.

For Subgroup I-B the mean hearing level three months after the operation was 27.0 db and one year after the operation it was 29.4 db, a loss of 2.4 db. The mean hearing level for the last post-operative test of hearing (6.94 years post-operatively) was 29.0 db. The results of the statistical analysis of these data (see Appendix B, Table B-2) confirm our hypothesis that hearing does not get worse after the first year after the operation. This result confirms the conclusion of Davis, Walsh, and Eldert[7] that the decrement observed over the first year after the operation is related to the healing process.

Threshold Shift and Unreliability.

The results presented up to this point have dealt only with mean hearing levels at several post-operative intervals. Results stated in this form are of little use in evaluating individual cases. In order to present the information about the long-term stability of hearing in successfully fenestrated ears in a form more amenable to direct comparison with individual cases, we tabulated the amount and direction of threshold shift between one year after the operation and the last measure of hearing level (6.94 years) for the 113 cases in Subgroup I-B. These results are presented in Table III. In 64.7 per cent of the cases either there was no threshold

shift between one year after the operation and the last test of hearing, or the hearing level at the later date was better than it had been one year after the operation. The most improvement was 11 db and the largest loss was 15 db.

Thurlow *et al.*[5] have pointed out that there is random fluctuation involved in the measurement of the threshold for speech (spondaic words). If we use their estimates of the magnitude of this random fluctuation (standard error of measurement or measure of unreliability), we would expect

TABLE III.

Shift in Hearing Level Between Tests of Hearing One Year After Fenestration Operation and the Last Available Test of Hearing.*

Threshold Shift	Number	Per cent	Cumulative Per cent
9-11 db gain	1	.9	
7-8 db gain	5	4.4	5.3
5-6 db gain	11	13.3	18.6
3-4 db gain	16	14.2	32.8
1-2 db gain	23	20.4	53.2
0 db (no shift)	13	11.5	64.7
1-2 db loss	12	10.6	75.3
3-4 db loss	11	9.7	85.0
5-6 db loss	7	6.2	91.2
7-8 db loss	4	3.5	94.7
9-15 db loss	6	5.3	100.0
	113		

*The median post-operative interval for the last available test was 6.94 years.

about 33 per cent of the thresholds to have shifted more than 4.2 db, regardless of direction of shift. From Table III we can see that 38 out of the 113 thresholds, or 33.6 per cent, shifted more than 4.2 db. In addition, only 5 per cent of the thresholds would be expected to shift more than 8.4 db. Actually six of the 113, or 5.3 per cent, shifted more than 8.2 db. We conclude, on this basis, that the distribution of thresholds in Table III does not differ from what is predicted solely on the basis of random fluctuations involved in measuring the threshold for speech (spondaic words).

Analysis of data from Group I has shown, therefore, that there is a statistically significant loss of hearing during the first year after a successful fenestration operation, but that

there is no change in hearing level after the first year. The distribution of threshold shifts between 1.0 and 6.94 years (median post-operative interval for the last test of hearing) after the operation is not different from a theoretical distribution based on the random fluctuations involved in measuring hearing level for speech (spondaic words).

Stability of Hearing, Groups II and III.

Data were selected from Groups II and III to compare these groups to Group I with respect to the long-term stability of hearing in fenestrated ears. The data from Groups II and III do not cover as long a post-operative interval as those from Group I since these are chronological groups. The data for Groups II and III are more detailed for the first year after the operation, however, because a check-up six months after the operation was introduced into the schedule at the end of the period during which the cases in Group I were operated. In addition to comparing the groups with respect to long-term stability of hearing, we were able to determine whether the loss of hearing following fenestration took as long to stabilize in Groups II and III as it had taken in Group I.

Sixty-three patients from Group II, whose hearing level three months after the operation was 35 db or better returned for follow-up tests of hearing at six months, one year, and two years in addition to those at three months. Of these 63 patients, 29 had been operated early enough in the series to have returned for follow-up tests of hearing four years after the operation. The mean hearing levels of the 63 patients, Subgroup II-A, are plotted in Fig. 3 as a function of time after the operation. The mean hearing level for Subgroup II-B, 29 cases tested four years after the operation, is plotted at four years after the operation. The solid line in Fig. 3 is drawn to show that, as in the case of Group I, hearing gets worse during the first year after the operation but remains stable thereafter. Statistical analyses of the data for these subgroups (see Appendix B, Table B-3 and Table B-4) gave the same results that were obtained for Subgroup I-A (Appendix B, Table B-1).

In Group III, there were 42 patients whose hearing level

three months after the operation was 35 db or better, and who had returned for follow-up tests of hearing at six months, one year, and two years, as well as at three months after the operation. The mean levels for these 42 patients, Subgroup III-A, as a function of time after the operation, are plotted in Fig. 4, in which the solid line has been drawn to show that hearing level gets worse only during the first six months after the operation and then remains stable thereafter. The statistical analysis of these data (see Appendix B, Table B-5) indicates that the mean hearing level at six months after the operation is significantly worse than at three months after the operation, beyond the 1 per cent level of confidence, but that there are no differences among mean hearing levels for six months, one year, and two years.

Summary: Stability of Hearing.

In all three of these chronological groups the hearing level in fenestrated ears was found to be stable after an initial, statistically significant, loss of hearing. This threshold shift was 2.5 db for fenestrated ears in Groups I and II, but was only 1.4 db in Group III. For Groups I and II the shift was not complete until the end of the first year after the operation.

Improvement in the results of fenestration surgery in terms of final hearing level can also be demonstrated in terms of a decrease in the shift for the worse that immediately follows fenestration surgery and in terms of the time course of this shift. The threshold shift decreases from 2.5 db in Groups I and II to 1.4 db in Group III.

The only change in technique between the periods covered by Groups II and III was the introduction of Neo-Cortef ointment (Upjohn) in packs in the cavity and over the flap in June, 1953.

STABILITY OF HEARING IN UNOPERATED OTOSCLEROTIC EARS.

The purpose of this portion of the study is to determine the rate at which hearing loss increases in unoperated, otosclerotic ears over long periods of time.

Data were available on 34 patients from Group I (Sub-

group I-C), on whom only one ear had been operated. These patients had returned for follow-up tests on both their fenestrated and their unoperated ears one, three, and seven years, as well as at three months after the operation.

The mean hearing levels at the various intervals after the operation are plotted in Fig. 5. According to the statistical analysis of these data, the only statistically significant shift in threshold took place between the third and the seventh

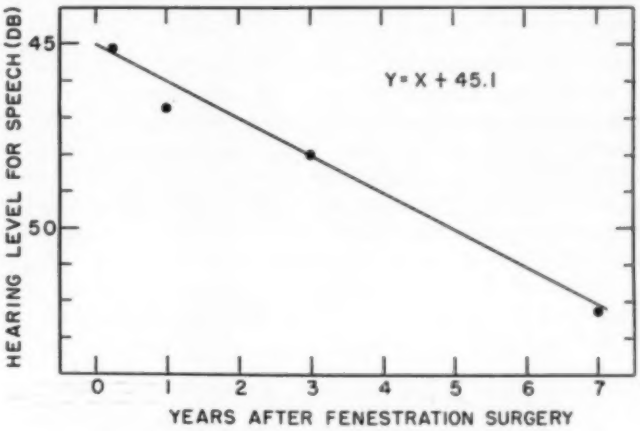

Fig. 5. Mean hearing level for speech (spondaic words) as a function of time after surgery was performed on the contralateral ear for 34 unoperated otosclerotic ears from Group I (Subgroup I-C). The line has been fitted to the data by the least squares technique.

year after the operation. The solid line drawn in Fig. 5 was fitted to the points by a least squares technique and this line, with a slope of 1 db per year, fits the data very well. Because the rate at which hearing level rises is so slow, 1 db per year, the statistical analysis could not detect the shift over only one or two years, but a linear trend is clearly demonstrated by these data as a whole.

The comparison of the stability of hearing level in fenestrated and unoperated otosclerotic ears reveals that while hearing level does not change appreciably in fenestrated ears

after the operation, except for the slight shift during the first year after operation, the unoperated otosclerotic ears very slowly become less sensitive, at a rate of about 1 db per year.

STABILITY OF SPEECH DISCRIMINATION SCORES IN FENESTRATED AND UNOPERATED OTOSCLEROTIC EARS.

The purpose of this part of the study is to determine whether the speech discrimination scores for fenestrated and unoperated otosclerotic ears remain stable over long periods of time.

There were 37 patients in Group I with one fenestrated and one unoperated otosclerotic ear. In this group of patients the hearing level in the fenestrated ear three months after the operation was 35 db or better. They had returned for follow-up tests of hearing on both ears at three months, three years, and seven years. The mean age of these patients at the time the operation was performed was 33.7 years. Speech discrimination scores were measured with the Rush Hughes recordings of the PB word lists. These recordings were played back to the patient at sound pressure levels of at least 100 db (re 0.0002 dynes/cm^2).

The mean speech discrimination scores for the fenestrated and for the unoperated ears of these patients are plotted in Fig. 6 as a function of time after the operation. A single line has been drawn in Fig. 6 to show that no statistically significant differences were found between the mean speech discrimination scores for the fenestrated and for the unoperated otosclerotic ears at any interval after the operation (see Appendix D, Table D-1). Both ears, however, show a decrease in speech discrimination scores between the third and seventh year after the operation that is statistically significant at the 1 per cent level of confidence.

It has been claimed that the fenestration operation will prevent the development of secondary nerve deafness. Because the maximum discrimination score can be assumed to measure the integrity of the cochlea,[1] we are able to evaluate this claim for a seven-year period of time. The result, no

difference between the fenestrated and the unoperated ear, three or seven years after surgery, fails to support this claim.

The observed decrease in speech discrimination scores of 5.3 per cent between the third and seventh year after the operation can be explained in either of two ways. First, it is possible that this decrease is a result of the continuing oto-

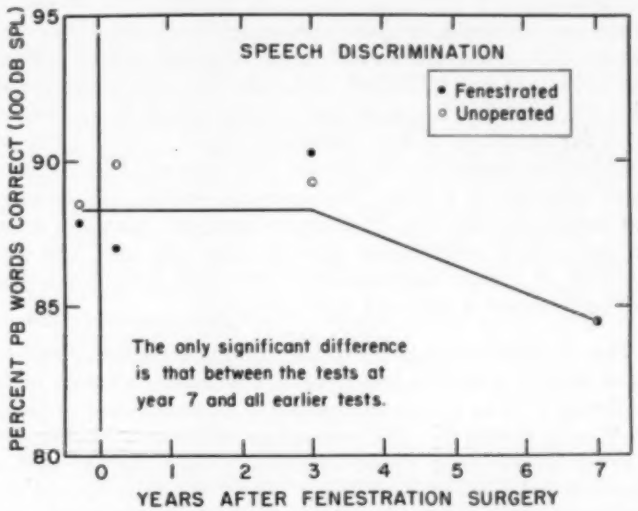

Fig. 6. Speech discrimination in both fenestrated and unoperated ears as a function of time after surgery for 37 patients from Group I (Subgroup I-D). The line represents an interpretation of the statistical analysis and is not fitted to the data.

sclerotic process, and that this process is not impeded by the fenestration operation. On the other hand, it is possible that a comparable loss of speech discrimination will occur in a similar group of normal ears between the ages of 36.7 and 40.7 years as a result of aging. In order to determine which of these explanations is the most valid, it would be necessary to measure the speech discrimination of a group of people with normal ears at comparable age intervals. If the people with normal ears show a decrease in speech discrimination

score of 5 per cent between the ages of 36.7 and 40.7 years, we could assume that the decrease observed here was the result of normal aging and not the result of the otosclerosis.

SUMMARY.

Follow-up tests of hearing have been conducted over periods from three months to nine years on 818 patients upon whom the fenestration operation has been performed. These patients are divided into three chronological groups. The first group, Group I, includes all cases reported earlier (1951), and Groups II and III include 486 cases heretofore unreported. The three groups are compared with respect to, *1.* hearing level for spondaic words measured three months after the fenestration operation; *2.* closure of the fenestra, and *3.* long-term stability of hearing for spondaic words. In addition, the long-term stability of hearing level for spondaic words in unoperated otosclerotic ears is assessed, and long-term stability of speech discrimination scores in both fenestrated and unoperated otosclerotic ears is studied.

The average hearing level for spondaic words, measured three months after the fenestration operation, is 3.5 db better in Group II than in Group I, and 3.1 db better still in Group III than in Group II. The percentage of patients having final hearing levels of 20 db or better was only 3.0 per cent in Group I, but increased to 12.1 per cent in Group II and 28.2 per cent in Group III. The percentage of patients with final hearing levels worse than 35 db decreased from 27.6 per cent in Group I to 16.8 per cent in Group II to only 9.2 per cent in Group III.

Closure occurred in 14.6 per cent of the cases in Group I, but in only 2.8 per cent of Group II and 3.3 per cent of Group III.

Hearing level for spondaic words in fenestrated ears remained stable after an initial loss. In Groups I and II this early threshold shift was 2.5 db and took place during the first year after surgery. In Group III the initial loss of sensitivity was only 1.4 db and was complete at the end of six months.

Hearing in unoperated otosclerotic ears got worse at a rate of about 1 db per year, when time was counted from the date at which the fenestration operation was performed on the opposite ear.

Speech discrimination scores were the same in 37 unoperated otosclerotic ears as they were in the corresponding fenestrated ears. The speech discrimination scores did *not* decrease more rapidly in the unoperated than in the fenestrated ear. Between the third and seventh postoperative year, speech discrimination scores for both ears decreased by about 5 per cent.

In general, after a fenestration operation we may expect the final hearing level in the fenestrated ear to increase slightly in the first six months after the operation and to remain stable thereafter. In the unoperated otosclerotic ear, on the other hand, hearing for spondaic words will get worse at the rate of about 1 db each year after the time of the operation. The fenestration operation does not affect the slight fall in speech discrimination scores which occurs over a period of several years.

APPENDIX A.

The first step in the comparison among the three groups (I, II, and III) was to perform an analysis of variance on the pre-operative hearing levels. The analysis of variance design used was a simple, single classification analysis of variance.[13] The assumption of homogeneity of variance involved in this analysis was tested (Bartlett's test) and found to be tenable.

The results of the analysis of variance performed on the pre-operative hearing levels are presented in Table A-1. The significant "among groups" effect indicates that the three groups were different with respect to pre-operative hearing level. This result indicates that the appropriate manner of comparing the groups with respect to the outcome of the fenestration operation is the technique of analysis of covariance. This technique enables us to "match" the groups with respect to pre-operative hearing levels.

The analysis of covariance technique involves a complicated set of assumptions that must be tested empirically as part of the computational procedure. These assumptions deal with the correlation between pre-operative and final hearing level in the present case. These assumptions can be summarized by saying that it is necessary to show that there is a significant correlation between pre-operative and final hearing

TABLE A-1.

Results of Analysis of Variance: Comparison Among Groups I, II, and III With Respect to Pre-Operative Hearing Level.

Source of Variation	df	Mean Square	F
Among Groups I, II, and III	3	201.65	4.33*
Error	655	46.59	

*Significant beyond the 1 per cent level of confidence.

TABLE A-2.

Results of Analysis of Covariance: Comparison Among Groups I, II, and III with Respect to Final Hearing Level.

Source of Variation	df	Adjusted Mean Square	F
Among Groups I, II, and III	3	2,314.23	23.97*
Error	654	96.55	

*Significant beyond the 1 per cent level of confidence.

level, and that this correlation is essentially the same in every group. The assumptions were tested and found to be tenable.

The results of the analysis of covariance performed on the final hearing levels are presented in Table A-2. These results indicate that there was, on the average, an improvement of 3.25 db between successive groups, and that this improvement was highly significant. The degree of correlation between pre-operative and final hearing level is .34 and it, too, is highly significant.

FINAL HEARING LEVEL VS. GAIN.

In applied statistics the analysis of covariance is always used instead of a difference (between pre-operative and final hearing level) score, because the conditions that must be satisfied before gain (difference) can be used legitimately

are very difficult to fulfill in a non-laboratory context. This comment is appropriate, since the issue of final hearing level vs. gain is relevant to otological surgery. Although Davis and Walsh[7] have already discussed this issue in terms of the limits of improvement in hearing following fenestration surgery, it still remains open. We shall discuss the issue from the point of view of the logic relating to statistics and measurement, but we shall not ignore the point of view presented by Davis and Walsh.

Any measure of "gain" is suspect in statistics because its use assumes that the amount of gain is independent of the initial measure (pre-operative hearing level). This assumption cannot be met in the case of fenestration surgery for two reasons: first, there is a real limit on how efficiently an ear can function once the ossicular chain has been interrupted. Davis and Walsh[7] point out that because of this limitation, final hearing levels cannot be much better than 20 db. In addition, the fenestration operation can only remedy the conductive component of a hearing loss. Since the maximum conductive component of a hearing loss is limited to about 60 db (because of bone conduction), the maximum possible improvement is about 40 db (60 db − 20 db = 40 db).[7]

Whenever a large number of cases is distributed within limits such as those described by Davis and Walsh[7] (discussed above), we must expect a significant correlation to appear. In the present study, the correlation between pre-operative and final hearing level of .34 was used to facilitate the comparison among groups with respect to final hearing level, but the correlation is not large enough to use in predicting the outcome of any individual operations, since only 11.4 per cent of the variability in final hearing level can be accounted for by the correlation between pre-operative and final hearing level. This leaves us with Final Hearing Level as the measure to use in evaluating the improvement in hearing following a fenestration operation. *The best prediction that a surgeon can make is that each case of purely conductive hearing loss will reach the mean final hearing level for his previous patients.*

APPENDIX B.

The analysis of variance design used to test the stability of hearing in fenestrated ears as a function of years after the fenestration operation, was a simple two-way classification design in which patients were assumed to be a random variable.[13] The same design was used on all five subgroups. As shown in Tables B-1 to B-5, a statistically significant dif-

TABLE B-1.

Results of Analysis of Variance: Stability of Hearing in Fenestrated Ears. (Subgroup I-A, N = 43).

Source of Variation	df	Mean Square	F
Patients	42	109.75	
Years after surgery	3	89.43	6.45*
Error	126	13.86	

*Significant beyond the 1 per cent level of confidence.

TABLE B-2.

Results of Analysis of Variance: Stability of Hearing in Fenestrated Ears. (Subgroup I-B, N = 113).

Source of Variation	df	Mean Square	F
Patients	112	74.98	
Years after surgery	2	193.18	14.24*
Error	224	13.57	

*Significant beyond the 1 per cent level of confidence.

TABLE B-3.

Results of Analysis of Variance: Stability of Hearing in Fenestrated Ears. (Subgroup II-A, N = 63).

Source of Variation	df	Mean Square	F
Patients	62	209.44	
Years after surgery	3	116.12	3.76*
Error	186	30.92	

*Significant beyond the 1 per cent level of confidence.

TABLE B-4.

Results of Analysis of Variance: Stability of Hearing in Fenestrated Ears. (Subgroup II-B, N = 29).

Source of Variation	df	Mean Square	F
Patients	28	150.59	
Years after surgery	4	63.08	6.08*
Error	112	10.37	

*Significant beyond the 1 per cent level of confidence.

TABLE B-5.

Results of Analysis of Variance: Stability of Hearing in Fenestrated Ears. (Subgroup III-A, N = 42).

Source of Variation	df	Mean Square	F
Patients	41	68.62	
Years after surgery	3	31.45	4.90*
Error	123	6.42	

*Significant beyond the 1 per cent level of confidence.

ference was found "among years after surgery" for each of the five subgroups. The Newman-Keuls sequential range test[14] was used to determine the significance of the differences among the various individual means. The outcome of the comparisons among the individual mean hearing levels as a function of time after fenestration surgery is described in the text for each subgroup.

APPENDIX C.

The analysis of variance design used to test for the stability of hearing in the unoperated otosclerotic ears was again a simple two-way classification analysis of variance in which patients were assumed to be a random variable.[13] A statistically significant difference among "years after surgery" was found. The sequential range test[14] indicated that the

TABLE C-1.

Results of Analysis of Variance: Stability of Hearing in Unoperated Ears. (Subgroup I-C, N = 34).

Source of Variation	df	Mean Square	F
Patients	33	225.13	
Years after surgery†	3	322.30	11.02*
Error	99	29.26	

*Significant beyond the 1 per cent level of confidence.
†Surgery performed on contralateral ear.

only significant difference among the mean hearing levels for years after surgery was between years three and seven. The line drawn in Fig. 5 was fitted by least squares. The fit is good enough to allow us to assume that the change over one or two years is too small to be detected.

APPENDIX D.

The analysis of variance design used to test for difference between the operated and unoperated ears as a function of time after surgery was a three-way classification analysis of variance in which patients were assumed to be a random variable.[13] The percentage scores on the PB words were converted to the arcsin of the square root of the percentage to meet the assumption of homogeneity of variance,[13]

TABLE D-1.

Results of Analysis of Variance: Stability of Speech Discrimination Scores for Fenestrated and Unoperated Ears. (Subgroup I-D, N = 37).

Source of Variation	df	Mean Square	F
Patients (P)	36	.2065	
Fenestrated and unoperated ears (E)	1	.0467	.98
Interaction of E and P	36	.0477	
Years after surgery (Y)	3	.4107	7.66*
Interaction of Y and P	108	.0536	
Interaction of E and Y	3	.0402	1.93
Interaction of E, Y, and P	108	.0208	

*Significant beyond the 1 per cent level of confidence.

and the analysis was performed on these transformed scores. The failure of the interaction between years and operated vs. unoperated ears is the most important result in Table D-1. The interpretation of this result is discussed in the text. The sequential range test[14] was used to compare the mean PB scores at the different post-operative intervals, and only the difference between years three and seven was found to be significant.

REFERENCES.

1. Walsh, T. E., and Silverman, S. R.: Diagnosis and Evaluation of Fenestration. The Laryngoscope, 56:536-555, 1946.

2. Falconer, G. A., and Davis, H.: The Intelligibility of Connected Discourse as a Test for the "Threshold for Speech." The Laryngoscope, 57:581-595, 1947.

3. Thurlow, W. R.; Silverman, S. R.; Davis, H., and Walsh, T. E.: A Statistical Study of Auditory Tests in Relation to the Fenestration Operation. The Laryngoscope, 58:43-66, 1948.

4. Silverman, S. R.; Thurlow, W. R.; Walsh, T. E., and Davis, H.: Improvement in the Social Adequacy of Hearing Following the Fenestration Operation. The Laryngoscope, 58:607-631, 1948.

5. THURLOW, W. R.; SILVERMAN, S. R.; DAVIS, H., and WALSH, T. E.: Further Statistical Study of Auditory Tests in Relation to the Fenestration Operation. THE LARYNGOSCOPE, 59:113-129, 1949.

6. DAVIS, H., and WALSH, T. E.: The Limits of Improvement of Hearing Following the Fenestration Operation. THE LARYNGOSCOPE, 60:273-295, 1950.

7. DAVIS, H.; WALSH, T. E., and ELDERT, E.: Closure and Revision Following the Fenestration Operation. THE LARYNGOSCOPE, 60:1181-1189, 1950.

8. THURLOW, W. R.; DAVIS, H.; WALSH, T. E., and ELDERT, E.: A Third Statistical Study of Auditory Tests in Relation to the Fenestration Operation. THE LARYNGOSCOPE, 61:124-137, 1951.

9. ELDERT, E., and DAVIS, H.: The Articulation Function of Patients with Conductive Deafness. THE LARYNGOSCOPE, 61:891-909, 1951.

10. HIRSH, I. J.; DAVIS, H.; SILVERMAN, S. R.; ELDERT, E. G., and BENSON, R. W.: Development of Materials for Speech Audiometry. *Jour. Speech, Hear. Disord.*, 17:321-337, 1952.

11. SILVERMAN, S. R., and HIRSH, I. J.: Problems Related to the Use of Speech in Clinical Audiometry. *Ann. Otol., Rhinol. and Laryngol.*, 64:1234-1244, 1955.

12. DAVIS, H.; HOOPLE, G., and PARRACK, H. O.: The Medical Principles of Monitoring Audiometry. *A.M.A. Arch. Indust. Med.*, 17:1-20, 1958.

13. WALKER, H., and LEV, J.: "Statistical Inference," pp. 348-425, Henry Holt & Co., New York, 1953.

14. DUNCAN, D. B.: Multiple Range and Multiple F Tests. *Biometrics*, 11:1-42, 1955.

NEW RESEARCH LABORATORY.

The Elbyrne G. Gill Eye and Ear Foundation announces the establishment of a Research Laboratory in conjunction with the Eye Bank and Sight Conservation Society of Virginia under the direction of Miss Jean Swartz, M.S. in chemistry. The laboratory is prepared to make the dye test for Toxoplasmosis.

FURTHER STUDIES ON THE PATHOLOGY OF OSSICLES IN OTOSCLEROSIS.*

W. P. COVELL, M.D.,
and
M. FEINMESSER, M.D.,†
St. Louis, Mo.

A large collection of sectioned and stained preparations of the incus and head of the malleus has been made available for study as a result of fenestration surgery. The bone and joint changes in similar material have been described by Lempert and Wolf.[1,2] Other observations on ossicles in place in sectioned temporal bones, showing otosclerotic foci with and without ankylosis of the footplate of the stapes, have been reported (Covell[3]). From studies up to the present time it has become apparent that:

1. Alignment of the ossicular chain is frequently altered and individual ossicles of the chain may become distorted in shape;

2. Changes occur within the articular cartilages, fibrocartilaginous pad and capsules of the joints of an atrophic and degenerative nature;

3. The bone, chiefly in the region of the articular surface, usually shows irregularity in pattern; large canals, altered vascularity, blue mantles, fibrous marrow, lipping, and an occasional hyperostosis may occur; and,

4. Similar bony changes without joint pathology may occur in the absence of ankylosis, but in the presence of an otosclerotic focus within the otic capsule.

The purpose of the present studies is three-fold: first, to

*From the Department of Otolaryngology, Washington University School of Medicine, St. Louis, Mo.

†Of the Ear, Nose and Throat Department, Rothschild-Hadassah-University Hospital, Jerusalem, Israel, and a visiting research fellow at Central Institute for the Deaf, 1957-1958.

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication May 15, 1958.

compare the incidence of bone and joint changes for this material with those of previous observations;[1,2] second, to compare the bony changes with those described by Nager and Ruedi,[4] and Ogilvie and Hall[5] in the small piece of bone consisting of three layers of the otic capsule removed at the time of fenestration from the roof of the ampullary end of the horizontal canal; each of these reports described bony changes in 13 per cent of the specimens; these percentages are in close agreement to those Nylen,[6] who studied sections of temporal bones from otosclerotics and reported an incidence of alterations in this area for 15 per cent of the specimens; third, to review the structural differences between the bone

TABLE I.

Changes Within the Incus, Head of the Malleus and Malleo-Incudal Joint Surfaces Classified According to Degrees of Severity. (Percentage Values—209 Specimens).

	Malleus Head		Incus		Diffuse Bony Changes (%)		Joint Surfaces	
	No.	%	No.	%	Malleus	Incus	No.	%
Group I	(79)	37.8	(98)	46.9	10.1	7.1	104	49.7
Group II	(110)	52.6	(95)	45.5	20.9	13.7	84	40.2
Group III	(16)	7.7	(13)	6.2	56.3	46.2	17	8.1
Group IV	(4)	1.9	(3)	1.4	75.0	53.4	4	2.0

of the ossicles and that of the capsule in terms of the pathology of the lesions; a feature common to each is the presence of calcified cartilaginous rests. Typical otosclerotic foci within the ossicles are known to be a rarity,[3,7] and probably occur in only the most "malignant" form of the disease.

The present report is to be considered as a preliminary study, since a subsequent histochemical analysis of bone of the ossicles and otic capsule may yield more pertinent information.

RESULTS.

A classification (see Table I) of changes in bone of ossicles and in joint surfaces was made on the following basis: Group I—the bone showed no changes, or only slight alterations such as early blue mantles and/or beginning canalization; the joint surfaces were without changes or revealed evidence

of a beginning necrosis of articular cartilage in the area adjacent to bone of the ossicle. Group II—alterations in the bone were considered to be of mild to moderate degree (see Fig. 1); these consisted of prominent blue mantles, beginning irregularity in formation of new bone; large canals, some containing loose fibrous connective tissue and blood vessels; the joint changes were considered to be thinning and necrosis

Fig. 1. An early change in bone of the head of the malleus showing some increase in diameter of canals that contain loose fibrous connective tissue and vessels. The bony wall of each canal has a narrow margin of deeply stained material. This particular specimen was classified in Group II. CCR, calcified cartilaginous rests; IR, irregularity in bony pattern. 72X.

of the basal portion of the articular cartilage, and occasionally irregular areas of overgrowth of cartilage in this layer. Group III—revealed moderate to severe changes such as marked irregularity in pattern of bone, remodeling of Haversian systems, prominent canalization and sometimes large fibrous-filled cavities; the articular cartilages showed erosion (see Fig. 3), calcification of the fibro-cartilaginous pad, and usually lipping of bone at the periphery of the joint. Group IV—changes (see Fig. 2) were extremely marked with replacement of old bone by fibrous connective tissue with poor vascularity, marked osteoporosis or an extreme amount of an

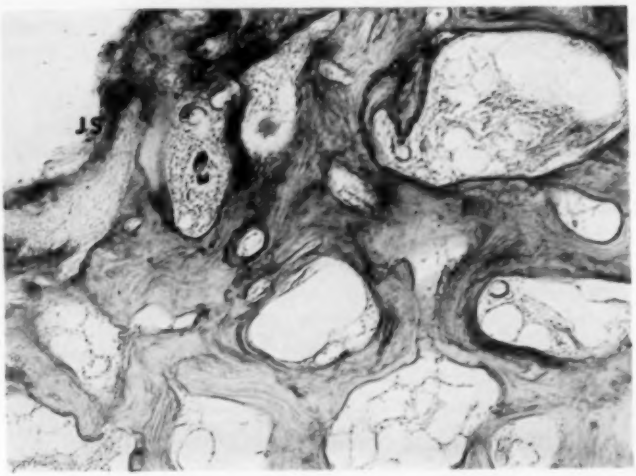

Fig. 2. An advanced type of change in the incus classified as Group IV. There is a marked osteoporosis with some fibrous marrow and erosion of the joint surface (JS) which showed ankylosis at one point. The greatest irregularity in the bony structure, aside from extreme canalization, occurred near the joint surface. 72X.

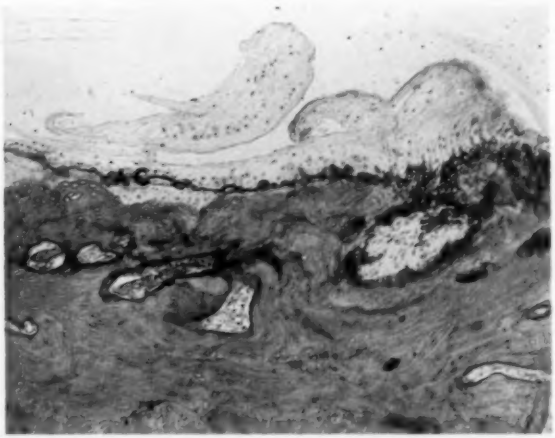

Fig. 3. Joint surface of the malleus showing erosion through the more basal layers of the articular cartilage (AC).

irregularly patterned bone; the joint cartilages were markedly eroded (see Fig. 2), with presence of some bony ankylosis.

At the time of making observations it was found that the bone of each ossicle nearest the joint surface revealed the more pronounced changes; however diffuse bony alterations were sometimes present. The percentages for diffuse involvement on the basis of classification into Groups are shown in Table I. Approximately 10 per cent of Group I showed diffuse changes in head and neck of the malleus and 7 per cent in the body and short process of the incus; the bone of the long process being less often involved. The percentage of specimens showing diffuse changes usually increased with the severity of the lesions, and were greater for the malleus than for the incus. Seventy-five per cent of Group IV revealed a diffuse type of change in the malleus, and 53.4 per cent in the incus.

In Table I are shown the total number of observations for the malleus head and whole incus with percentage values listed for each of the four groups.

Groups I and II were further subdivided into two groups each, depending upon the severity of the findings. Approximately 38 per cent of the preparations of the head of the malleus were Group I findings. Included under Group I are the specimens for which no bone or joint pathology was discernible. This amounted to 14, or 6.7 per cent of the 209 preparations of the head and neck of the malleus, and 25, or approximately 12 per cent of the incus specimens. The articular cartilages of the joints revealed no specific alterations in 31, or 14.8 per cent of the preparations. About 53 per cent of the malleolar findings fell into Group II. Twenty-two per cent of these were more marked in appearance than the remaining 78 per cent. There was 7.7 per cent of the 209 specimens in Group III, and 1.9 per cent in Group IV.

The results for incus studies were in about the same proportion to each malleus group, except there was a higher percentage showing Group I changes and a lower percentage showing Group II changes. The subdivisions for each group

were similar in percentage values to those for the malleus. In other words, 30 per cent of Group I were negative for findings, while 70 per cent definitely showed early changes. The percentages for Group III and IV were somewhat less, although comparable to those for the malleus.

Approximately 38 per cent of the preparations showed changes in the ossicles of a diffuse type. These were more numerous for the malleus than incus in the ratio of about 2:1.

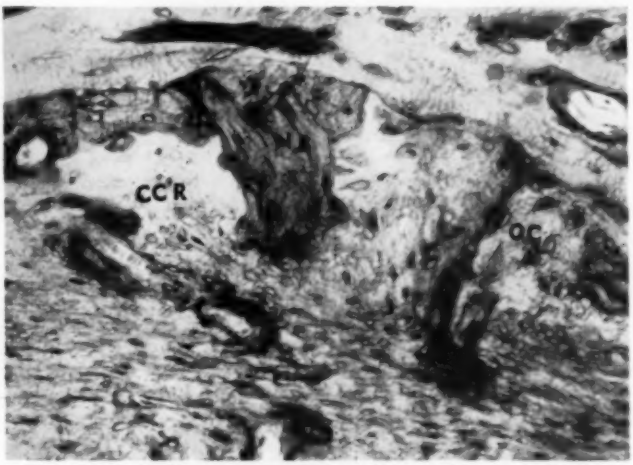

Fig. 4. Disintegration of calcified cartilaginous rests with an attempt to fill in the cavity with new lamellar-type bone. The osteocytes to the right are particularly irregular in size and shape, and their processes shrunken and distorted. CCR, calcified cartilaginous rests; NB, new bone formation; OC, osteocytes. 240X.

The changes in malleo-incudal articular cartilages, also classified according to four groups, were approximately 49.7 per cent, Group I; 40.2 per cent, Group II; 8.1 per cent, Group III, and 2 per cent, Group IV. The changes in the joint surfaces were considered to be less marked than those in the ossicles in 20 per cent of the specimens, and of approximately the same value for changes more marked in the joints than those in the ossicles. The grouping of the joint changes and those for the bone were usually in close agreement. When a

difference was present it was usually of the next, or preceding group. In other words, a Group I bony change was never accompanied by Group IV joint change, and in only one instance was Group III joint change found for ossicles with Group I bony changes. It is possible that with the joint intact, additional findings might have been apparent and the classification somewhat different.

There remains only a few points to contribute to the histological detail of the alterations previously described.[1,2,3,4,5] Figure 4 represents a degenerative change within the calcified cartilaginous rests and an attempt to fill in the cavity with new bone. These calcified cartilaginous cells are apparently incapable of proliferation and formation of new cartilage. Vascular channels may also encroach upon such cavities, and they may become filled with a dense fibrous connective tissue. The distorted osteocytes (see Fig. 4) and the accompanying irregularity of pattern of bone produce a peculiar appearance. This has been described as "seedy" or "peppery."

DISCUSSION.

The present studies are in agreement with the histopathological findings previously described, and are similar to the changes in some of the specimens described by Ogilvie and Hall[5] for the small piece of bone removed from the ampullary end of the horizontal canal. The presence of canalization in the bone nearest the joints is one of the earliest changes. This is preceded and accompanied by the appearance of blue mantles, which are to be regarded as areas of absorption and deposition of bone. These and subsequent alterations occasionally become diffuse through most of the malleus and incus. The changes in the blood vessels and the resulting pathology in vascularity of the bone of these ossicles have been amply described by Lempert and Wolff.[1,2] The variations in the appearance of bone suggest an interference with nutrition and an abnormal metabolism.

The vascularity of the enchondral bone of the capsule and of the ossicles may be inadequate under certain conditions, because the proper nutrition of bone has been found to be dependent upon diffusion of substances from capillaries placed

within "a fraction of a millimeter of each bone cell."[7] The distorted and atrophic processes of osteocytes frequently seen in the vicinity of ossicular lesions would tend to support this view. It is undoubtedly the inherent low vitality and metabolism of enchondral bone that makes it particularly susceptible to any changes that might result from a diminished vascularity. According to Nager and Nager[8] the malleus and incus each contain a large cavity which is occupied by a "vascular convolute." The principal blood supply to these ossicles is by the "ossicular artery" which is a branch of the anterior tympanic artery. It divides into malleolar and incudal branches. While they described anastomoses between branches of the arteries, the existence of end arteries in the finer ramifications of the vessels cannot be refuted. The irregularity present in the cement lines of the laminated bone in some Haversian systems indicates periods of arrested growth. The present material was not stained by methods to demonstrate fibrils, but there is no doubt that woven and laminated areas would appear similar to those described for the otic capsule.[5] In the ossicles the mosaic pattern of the otosclerotic focus in the capsule is not predominate. There is, however, a tendency for irregularity of pattern and "seedy" appearance to be more pronounced in some specimens than others. The sparsity of osteoclasts in areas in which absorption of bone is prominent is not surprising, if we are to share the views of Harris and Ham[7] regarding their significance.

The bone that comprises the ossicles usually does not contain as many calcified cartilagenous rests as the enchondral layer of the otic capsule. Except for the compactness of this bone and its relatively poor vascularity the occurrence of calcified cartilaginous rests within it serve to distinguish it from most other skeletal bone. Any significance of the interglobular spaces and their contents in pathologic changes is uncertain, although they become encroached upon by disease and may disappear entirely. It is possible that present day histochemical and autoradiographic techniques might throw some light upon their importance.

The changes in the joint surfaces observed for ossicles from otosclerotics can be explained on the basis of *1.* altered

nutrition due to the vascular changes in the region of the joint surfaces. The first evidence of such is a necrosis of the basal layer of the articular cartilage which is comprised of large cartilage cells and clear matrix. Invariably the bone adjoining these areas showing this change exhibits the typical vascular alterations already described. The final result produces a marked thinning of the articular cartilage and irregularities in its thickness and composition in other areas. Occasionally the basal layer of the articular cartilage may undergo a compensatory overgrowth. Cavities filled with calcifying cartilage may be seen to encroach on the bone and later undergo necrosis. *2.* The joints between malleus and incus and incus and stapes reflect the ankylosis of the footplate of the stapes, by the appearances of calcium placques, uneven wearing of the articular disc and finally erosion of the articular cartilage with malposition of the joint surfaces, and even some degree of locking if not ankylosis of the malleo-incudal joint. Distortion of the joint surfaces may result in some displacement without ankylosis of the stapes footplate as shown in results reported by one of us.[3]

SUMMARY.

A classification of bony changes into four groups for 209 preparations, consisting of head and neck of malleus and the incus made available by fenestration surgery, revealed the following: *1.* approximately 38 per cent of malleus specimens showed none or only very slight to moderate changes, 7.7 per cent were considered as moderate to severe and 1.9 per cent as extreme; *2.* approximately 47 per cent of incus specimens were of Group I; 45.5 per cent, Group II; 6.2 per cent, Group III and 1.4 per cent, Group IV. The stained sections of the ossicles were further grouped according to diffuse or localized bony changes with the result that the more marked and severe changes were more often accompanied by diffuse bony changes.

Degenerative changes within the articular cartilages, erosion and even ankylosis of the malleo-incudal joint were found to occur in 50.3 per cent, while the remaining 49.7 per cent showed none or very slight changes.

REFERENCES.

1. Lempert, J., and Wolff, D.: Histopathology of the Incus and the Head of the Malleus in Cases of Stapedial Ankylosis. *Arch. Otolaryngol.*, 42:339, 1945.

2. Lempert, J., and Wolff, D.: Otosclerosis; Theory of Its Origin and Development. *Arch. Otolaryngol.*, 50:115-155, 1949.

3. Covell, W. P.: The Ossicles in Otosclerosis. *Acta Otolaryngol.*, 28:263-276, 1940.

4. Nager, F. R., and Ruedi, S.: Histologische Untersuchung der Fenestrations-splitter bei der Otoscleroseoperation. *Acta Otolaryngol.*, 39:300-313, 1951.

5. Ogilvie, R. F., and Hall, I. S.: Observations on the Pathology of Otosclerosis. *Jour. Laryngol. and Otol.*, 67:497-535, 1953.

6. Nylén, B.: Histopathological Investigations on the Localization, Number, Activity and Extent of Otosclerotic Focci. *Uppsala Läk*, Fören. Förh., 54:1-2, 1949.

7. Harris, W. R., and Ham, A. W.: The Mechanism of Nutrition in Bone and How It Affects Its Structure, Repair and Fate on Transplantation. Bone Structure and Metabolism, *Ciba Foundation* Symposium, pp. 135-147, 1956.

8. Nager, G. T., and Nager, M.: The Arteries of the Human Middle Ear, with Particular Regard to the Blood Supply of the Auditory Ossicles. *Ann. Otol., Rhinol. and Laryngol.*, 62:923-949, 1953.

ANNOUNCEMENT.

The Annual Spring Meeting of the Alumni Association of the New York Eye and Ear Infirmary will take place April 20-23, 1959, at the Infirmary.

Symposia will be offered on Headache; Allergy and Immunology in Otolaryngology; Diagnosis and Treatment of Laryngeal Conditions; Hearing Rehabilitation; and Rhinoplasty. In addition, there will be a closed circuit television demonstration of Head and Neck Surgery.

Additional information may be obtained by writing to Dr. John R. Finley, Secretary, Alumni Association, 218 Second Avenue, New York 3, N. Y.

LOUDNESS RECRUITMENT AND VERTIGO.*†

WALTER ALEXANDER, M.D.,

Winnipeg, Canada.

HISTORICAL.

Few sensations resulting from stimulation of special sense organs reach the conscious level from threshold intensities of stimulation. The ear is no exception; it rarely listens to sound of faint intensity, but is most frequently used to make intelligible the meaning of sounds which normally occur at 40 to 60 dbs above threshold. With the use of tuning forks, and for many years following the advent of the audiometer, the acuity of hearing was measured at minimal audibility, and little regard was paid to the ability of the patient to understand sound or speech at intensities well above threshold; indeed, at those levels where ordinary conversation is usually maintained.

Fowler[6] of New York described the "binaural loudness balance test" in 1936. He determined and recorded the phenomenon that some people had a difference in loudness intensity necessary for binaural loudness balance at different intensities, *i.e.*, a poor ear might hear loud sounds disproportionately well as compared with faint sounds. On the other hand, in some patients there was a constant difference or no difference in intensity necessary to balance, binaurally, the loudness at all frequencies. Fowler's findings, in essence, differentiated the normal and/or conductively deaf ear from those with perceptive hearing losses.

The binaural loudness balance test (to be described) did not engender great enthusiasm among otologists for some years; however, in the late 1940's Fowler's concepts were revived by British and European otologists, and in more re-

†Reprinted from Prooceedings of the Canadian Otolaryngological Society, 1957.

*Read at the meeting of the Canadian Otological Society, Banff, Alta., Canada, June 17, 1958.

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication Nov. 17, 1958.

cent years considerable enthusiasm for the test, with some modifications, has been in evidence in the American literature.

METHOD OF TESTING FOR RECRUITMENT.

The patient wears a set of earphones which are connected to a single audiometer with arrangements for independent adjustments of the intensities. The frequency of the sound is the same in each earphone, and the examiner may switch the sound alternately from the left to the right ear, or vice versa.

The threshold of hearing for either ear is determined conventionally and recorded on the audiograms. The diagrams in Fig. 1 represent the audiograms of a patient with conduction deafness, a normal hearing patient, and one with perception deafness.

The audiometer is a popular one, with two channels recording the hearing loss in the right or left ear independently or simultaneously. The binaural loudness balance test may be used at any frequency from 250 to 4000, but most frequently is recorded at the 1000 cycle frequency. A sound of given intensity, say 30 dbs, is delivered to the better ear, and the intensity of sound delivered to the poorer hearing ear is increased until the patient states that the intensity of the sound is equally loud in the two ears. The differential is recorded as indicated in the lower diagrams in Fig. 1. The sound intensity is then increased to 40 or 50 or 60 dbs in the better ear, and the intensity in the second channel delivering sound to the poorer ear is increased until the patient remarks that the sound is equally loud in either ear; the corresponding points of equal loudness are then recorded on the graph.

In a case of unilateral conductive deafness, the differential of sound intensities necessary to balance the sensation of loudness in either ear will be shifted the same number of decibels as is evident at threshold. In normal hearing ears, of course, the sound intensity necessary to create a sensation of equal loudness will always be of an equal number of decibels intensity in either ear. On the other hand, in some types of perceptive hearing loss, the differential which is found at

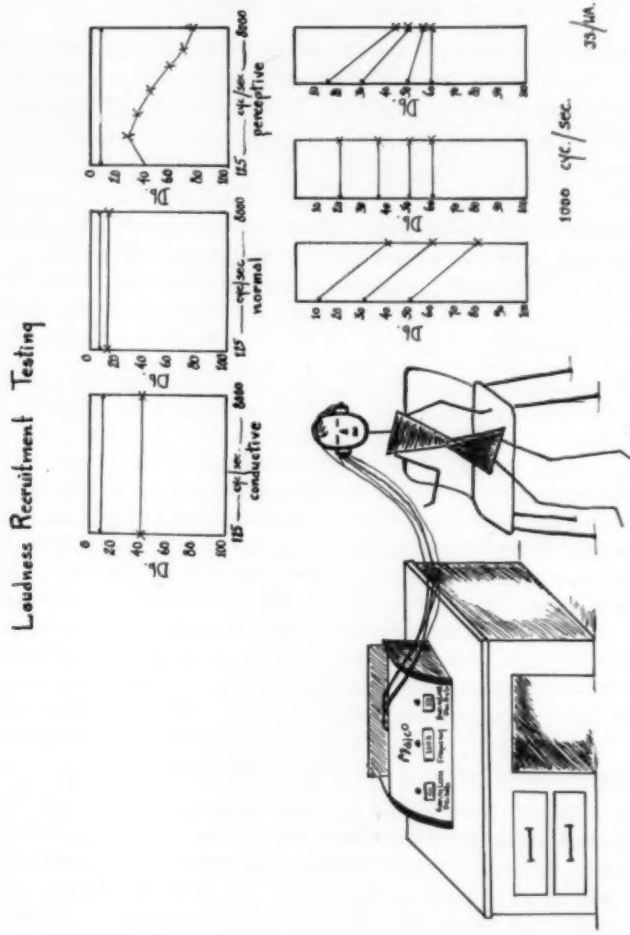
Loudness Recruitment Testing
conductive
normal
perceptive
DL
1000 cyc./sec.
33/μA.

Fig. 1.

threshold will gradually subside until at high intensities the sound pressures necessary to create a sensation of equal loudness in the two ears will parallel one another, even though a marked discrepancy may be present at threshold intensities. In some types of perceptive deafness, the deafness of the affected ear at threshold disappears at the higher intensities and this, in essence, constitutes the phenomenon of loudness recruitment as evidenced with the binaural loudness balance test of Fowler.

Because the physiological phenomenon of "adaptation" may influence the recorded end-point in recruitment testing, it is necessary to instruct the patient explicitly with regard to the end-point of balanced hearing sensation and thereafter carry out the test with dispatch.

Fowler's test for binaural loudness balance is easily and simply carried out; however, some modifications have been proposed which give quite comparable results. Such tests are known as the "bifrequency loudness balance test," the "de Bruines-Altes test," etc.

DIFFERENCE LIMEN TESTING.

Lüscher[19] states: "The difference limen for tone intensity variations is the smallest change of tone intensity, *i.e.*, the smallest variation of the sound pressure, which is just perceived as a variation of the subjective loudness, that is the absolute difference limen."

"The smallest detectable change in the intensity of a tone determines the intensity differential sensitivity of the ear." (Stevens and Davis.[23])

Brinitzer (1935), and Goren de Mare mentioned it in 1939, and Bekesy published his results in 1947. There has since developed a rather large number of variances in testing for the difference limen. Bekesy's recruitment test measures the intensity difference limen for loudness at the threshold of hearing. When the difference limen is smaller than normal, recruitment is present; when the difference limen is normal, recruitment is absent. There has been some criticism of the test, because it is carried out at threshold intensities and the

fatigue effect of listening to near-threshold sounds may alter the reaction time of the individual being tested.

A similar type of testing was developed by Lüscher and Zwislocki, in which the difference limina are tested at well above threshold, namely 40 dbs. At this level, the normal difference limen is practically independent of the frequency. The greatest degree of accuracy in this test is reached in the middle frequencies from 250 to 4000 cycles, and it is said to be less accurate below and above, especially in the upper tone limit.

In 1952, Jerger[10] modified the Lüscher-Zwislocki technique somewhat. As Bekesy and Reger believe that the greatest degree of recruitment occurs just at and above threshold, Jerger chose a test level close to threshold, namely 15 dbs above threshold, as that intensity level at which recruitment should be carried on. Another modification of the difference limen test was developed by Denes and Naunton.[1]

The difference limen tests have the advantage over the loudness balance test in that they can be carried out for monaural hearing losses.

Lüscher's paper was presented on the basis of the examination of 71 patients and he states: "The difference limen means a more rapid increase than normal in the subjective loudness when the tone intensity rises, so that in spite of deafness at threshold, loud tones are perceived as being equally loud as in normal hearing. The recruitment phenomenon is not in evidence in conductive deafness, and is characteristic of certain forms of perceptive deafness. Like the difference limen, it concerns the relationship between the increase of subjective loudness and the increase of tone intensity and, in theory, it may be concluded that the more rapid increase of subjective loudness presupposes a smaller difference limen. As the increase in subjective loudness occurs most rapidly immediately above threshold, the phenomenon must be in evidence, particularly at threshold and when the tone intensity is small . . . These theoretical considerations have led to the generally accepted opinion that the recruitment phenomenon and difference limen have the same significance and, further-

more, that the difference limen may be determined instead of the recruitment phenomenon, and, on examination in this instance of 15 patients of different degrees of deafness, have made it clear that the two phenomenona are parallel. There is not only fundamentally identical behavior, but also in detail they plot the same curve. This furnishes direct proof that the same significance is to be attached to the difference limen as to the recruitment phenomenon and, therefore, in cases of binaural deafness the determination of the recruitment phenomenon may be replaced entirely by that of the difference limen. In cases of monaural deafness the determination of the recruitment phenomenon is equivalent to that of the difference limen."

Jerger, in nine cases of sensori-neural loss, performed the difference limen test and the binaural loudness balance test, found that the tests yielded equivalent results, and concluded that abnormally small difference limens and the recruitment phenomenon parallel one another with fidelity. Neuberger tested 14 cases of unilateral hearing loss by both the Lüscher difference limen test and the alternate binaural loudness balance test and found complete parallelism between the recruitment phenomenon and the decreased difference limen.

SECTION OF THE VIIITH NERVE: HISTOLOGY OF THE ORGAN OF CORTI.

1. Wittmaack, in 1911, reported that the division of the cochlear nerve resulted in degeneration of the spiral ganglion, but that the organ of Corti remained normal.

2. Kaida, in 1931, and Hallpike and Rawdon-Smith, in 1934, confirmed these findings.

3. Dandy reported his findings of partial section of the VIIIth cranial nerve for the relief of Ménière's disease in 1934. It was reported that frequently no change in auditory threshold occurred, even though large portions of the nerve were sacrificed.

4. Schuknecht and Woellner,[21] in 1955, performed partial VIIIth nerve section on cats, and in all instances the histological sections of the organ of Corti were completely normal,

except in those cases in which the blood supply had been interfered with. In such cases, degenerative changes were observed in the organ of Corti.

TUMORS AND/OR PRESSURE ON THE VIIITH NERVE: EFFECTS ON THE ORGAN OF CORTI.

In VIIIth nerve tumors, the tumor fills the internal auditory meatus and causes a compression destruction of the fibers of the VIIIth nerve. There is degeneration of the spiral ganglion within the cochlea, but on the whole the organ of Corti is well preserved. These findings in human pathology substantiate the contentions referred to above, namely, that destruction of the VIIIth nerve by tumor or section does not destroy the organ of Corti *per se*, unless the blood supply is interfered with.

In Ménière's disease, or hydrops of the labyrinth, there is a dilatation of the cochlear duct. The cell mass of the organ of Corti is compressed and Corti's tunnel may be occupied by a structureless coagulum. In addition, the normal staining qualities of the cytoplasm and nuclei may be obliterated. Histological changes have not been apparent in the peripheral cochlear nerve fibers or in the cells of the spiral ganglion. Initially, the pathological process may be reversible and may account for the remissions which are so common in the symptomatology of the disease. With the evidence at hand, it seems reasonable to accept the fact that in hydrops of the labyrinth there are definite histological changes occurring in the endolymphatic system, the organ of Corti together with its hair cells, and it is at this site that the disturbance of hearing occurs.

ESTABLISHMENT OF THE SITE OF RECRUITMENT: COCHLEAR OR RETROCOCHLEAR.

Dix and Hallpike[4] investigated 30 cases of clinical Ménière's disease or hydrops of the labyrinth and found the recruitment phenomenon present in all cases. The same authors performed the loudness recruitment test in 20 cases of degeneration of the VIIIth nerve. Eleven of the cases were of proven neurofibroma and the remaining nine cases were of other

varieties of cerebello-pontine angle lesions involving VIIIth nerve function. Of the 20 cases with lesions confined in or about the VIIIth nerve, the recruitment phenomenon was totally absent in 14 and there was slight evidence of recruitment in the remaining six cases.

Eby and Williams, at the Mayo Clinic, investigated 32 cases of Ménière's disease and six with VIIIth nerve neurinomas or other cerebellopontine angle lesions, and their findings with the loudness recruitment tests were consistent with those described by Dix and Hallpike.

Lundborg,[16] using the Bekesy audiometer, investigated 26 cases of clinically diagnosed Ménière's disease and found complete recruitment present in 24 of the 26 cases. Accordingly, Lundborg's findings are in complete agreement with those of Dix, Hallpike and Hood, and Eby and Williams. Lundborg and his colleagues also investigated 21 cases of acoustic trauma, and in all cases the difference limen was strongly reduced, indicating complete recruitment at the frequencies where the hearing loss was evident on the threshold audiogram. Lundborg also recorded the findings in 21 cases of unilateral perceptive hearing loss which were due to retrocochlear lesions; in 18 of the 21 cases, there was complete absence of recruitment, and there was partial recruitment in the remaining three cases.

Jerger investigated 74 cases of sensori-neural losses and determined that there was a small difference limen in all cases investigated where the pathology could be determined or assumed to be in the end-organ, *i.e.*, the sensory receptor mechanism. The difference limina were found to be normal in those instances where the lesion was confined to the cerebellopontine angle.

Lüscher's findings indicated the absence of recruitment or normal difference limen in the presence of conductive deafness. Fourteen cases of acute or chronic acoustic trauma showed the characteristic results of a decrease in the difference limen or evidence of recruitment. Similar findings were recorded in six cases of clinical Ménière's disease. Difference limen was normal in five cases of retrolabyrinthine

hearing disorders due to cerebello-pontine angle tumors, streptomycin damage and, in one case, hearing loss following mumps.

Table I indicates the sex, and the site of the initial symptom, whether cochlear or vestibular, in 28 cases clinically diagnosed as Ménière's disease and in all of which the recruitment test was positive. With regard to sex, it would appear from our series that the disturbance is fairly evenly divided, with a slight preponderance in the males. It is interesting to note that in 12 of the 28 cases, the primary symptom was cochlear, either a loss of hearing or tinnitus. This symptom may in some cases precede by weeks or months the onset of vestibular symptoms. In only two of the 28 cases

TABLE I.—CLINICAL MENIERE'S DISEASE.

Sex	Onset of Symptoms: Primary Cochlear	Primary Vestibular	Primary Coch.-Vest.	Recruitment Ear
M-15	5	2	8	R-7 : L-8
F-13	7	0	5	R-5 : L-8

was the primary symptom vestibular without associated cochlear symptoms. In half of the cases, the symptomatology suggested that the cochlear and vestibular systems had been involved simultaneously.

PHYSIOLOGICAL AND ANATOMICAL CONCEPTS OF RECRUITMENT.

The loudness recruitment phenomenon has been established, on clinical grounds, as indicative of perceptive nerve deafness of the end-organ. It is, however, difficult to prove which histologic changes determine the presence or absence of the loudness recruitment phenomenon, because the end-organ and the auditory pathways have not been examined histologically in a sufficient number of instances, and only time and much hard work on the part of auditory histopathologists will clarify the problem.

Lorente de No explained the phenomenon in the following terms: "If a number of hair cells in the inner ear or a number of fibres in the cochlear nerve are missing, the tones will

appear to be weaker in intensity when near-threshold stimuli are used; but, if the intensity of the tone is increased, the more strongly activated hair cells or cochlear fibers will be sufficient to saturate, *i.e.*, to excite with the limiting intensity the cochlear fiber or the cells of the cochlear nuclei, so that the cerebral cortex will receive the same number of impulses per second from both ears and will perceive the tone delivered to the diseased ear as strongly as the tone delivered to the normal or less affected ear.

"Thus, it may be said that Fowler's phenomenon is an immediate consequence of the anatomy and physiology of the nervous system, and that, in fact, it must be pathognomonic of neural deafness."

Stevens and Davis[23] accept the view that the phenomenon of recruitment or, as they term it, the variable type of deafness, is attributable to a deficiency of neural elements. These authors report that a hearing loss caused by a masking sound would be of a variable type because the nerve fibers activated by the masking sound would be ineffective in contributing to the sensation of loudness of another tone heard in the presence of the masking noise. "Masking is an effective means of decreasing the available supply of neural elements and of producing the equivalent of a variable deafness." By this definition, variable deafness is tantamount to the presence of the recruitment phenomenon.

Lurie in 1940, as a result of brilliant experimental work on animals, established the fact that the outer hair cells are more prone to damage from toxins or from acoustic trauma than are the inner hair cells. The outer hair cells, of course, lie on the basilar membrane, whereas the inner cells lie directly against the bone. The former are presumably more easily damaged as the result of acoustic trauma. From a histological point of view, of course, it has been established by Lorente de No that each inner hair cell is supplied by a single nerve fiber, whereas a single nerve fiber has contact with many outer hair cells. Lurie conceived that the outer hair cells are sensitive to weak stimulation, whereas the inner cells exist for the perception of more intense stimulation. This concept

would mean that, with damage to the outer hair cells, the hearing for low intensities would be decreased; however, the presence of intact inner hair cells would presumably lead to a normal loudness sensation for high intensities. This could conceivably explain the loudness recruitment phenomenon on a histo-anatomical basis.

Patients suffering from acoustic trauma or Ménière's disease conceivably possess the ability of recruitment because weak sounds cannot stimulate the few remaining hair cells and only a minimum number of impulses will be conducted through the acoustic nerve. Strong stimulation, on the other hand, is able to stimulate the remaining intact hair cells (presumably inner hair cells) and the impulses are then conducted through the normal fibers of the acoustic nerve and its connections to the cerebral cortex, which then becomes relatively well saturated.

Lorente de No, in 1933, described at least a dozen anatomical regions within the cochlear nuclear mass. Each cochlear nerve fiber establishes contact with all regions and with many hundreds or thousands of cells. The impulses to the secondary auditory pathway are dependent upon the afferent cochlear impulses, and perhaps upon impulses which may be generated within the numerous cells constituting the cochlear nuclei. The multiple cellular cortical connections of the secondary auditory pathway allow complete saturation of this area if and when an impulse can be generated in the inner ear, presumably at the hair cell site. On the other hand, in retrocochlear lesions there is a relative decrease in the number of functioning nerve fibers, and a strong stimulus has no greater probability than a weaker one of reaching the cochlear nuclei or the auditory cortex. The hearing loss is, therefore, as great for weak as for strong sounds and the recruitment phenomenon cannot be elicited.

Tumarkin's explanation of loudness recruitment is not unlike that referred to above in the experimental work of Lurie. Tumarkin presumes that the basilar membrane is set into activity as the result of sound stimulation, and that the outer hair cells would be stimulated first and the inner hair cells later. If some of the hair cells are damaged, the pattern of

excitation will be that composed of the remaining hair cells. If, however, the intensity of a sound is increased, other viable hair cells may be brought into activity which in turn stimulate the intact acoustic nerve fibers and allow a comparatively greater number of impulses to reach the auditory area of the cortex. On the other hand, in deafness due to a disturbance of the acoustic nerve fibers the impulses initiated in the inner ear, likely in the region of the hair cells, are impeded to the same extent for both low and high intensities and because there are no available pathways; there will not, therefore, be any evidence of recruitment.

RELATIONSHIP OF RECRUITMENT PHENOMENON TO ADAPTATION.

Earlier in the paper it was remarked that perhaps some of the differences of opinion expressed with regard to the reliability of the difference limen test could be explained on the basis of a too prolonged period of testing, with resulting fatigue or adaptation of the nerve receptors.

The chief characteristic of fatigue in nerve tissue is the persistence of a reduced response following stimulation, and has its subjective counterpart in human experimentation in the so-called "post stimulatory fatigue." The amount or measure of fatigue is dependent upon the duration and intensity of the stimulation; as fatigue increases, the recovery time becomes longer.

"Adaptation" has been described as a process of equilibration—the maintenance of equilibrium between restoration and dissipation of energy. Davis states that: "It is a special case of fatigue, in that it represents an adjustment to a new level of sustained activity and not an exhaustion of reserve material." Equilibration occurs at practically all levels of stimulation and is a function of the frequency of stimulation but not its intensity. Hood[8] has indicated in the human subject that for intensities from 40 to 80 dbs above threshold, which cannot be considered supramaximal stimulation, adaptation reaches a maximum level at about the same time and is independent of the intensity of the test tone.

Hood summarizes the differentiation between post-stimula-

tory fatigue and adaptation as follows: "The one (fatigue) is a diminution of hearing acuity caused by a physical stimulus and varying in magnitude with its intensity, and the other (adaptation) is a decline in sensitivity of the hearing receptors consequent upon their physiological activity."

In disturbances of the end-organ of hearing the excitability of the end-organ to short intermittent stimulation may be normal or even increased. This "on-effect" is measured with the loudness recruitment or the difference limen test. On the other hand, in end-organ deafness the excitability of the nerve to sustained stimuli of even moderate intensities results in a relapse of excitability to a subnormal adapted level. It would appear, therefore, that subnormal adaptation to prolonged stimulation and the normal on-effect of excitability to brief intermittent stimulation are characteristic of end-organ deafness, and the two phenomena are likely closely related physiologically.

RELATIONSHIP OF RECRUITMENT TO SPEECH INTELLIGIBILITY.

It is generally agreed among most authors (Dix, Hallpike and Hood, 1943; Eby and Williams, 1951; Huizing and Reyntjes, 1952[9]) that in nerve deafness in which the recruitment phenomenon is present, speech intelligibility is decreased; as the loudness increases, intelligibility falls off, even with a very slight increase in intensity above threshold. On the other hand, in perceptive deafness other than end-organ deafness, intelligibility increases with intensity up to a certain level. One should not, of course, expect that every case of nerve fiber deafness will obtain improved intelligibility scores with amplification. There will presumably be cases in which the nerve fiber population is too small to carry a sufficient number of impulses towards the acoustic nuclei.

SUMMARY.

1. Section of the VIIIth nerve or acoustic tumor formation results in degeneration of the spiral ganglion, but the organ of Corti remains normal unless the blood supply is interfered with.

2. Ménière's disease connotes a pathological disturbance in the end-organ of hearing, *i.e.*, in the sensory receptor mechanism.

3. The binaural loudness balance test and the difference limen test yield equivalent results.

4. The recruitment phenomenon and/or decreased difference limen results from lesions in the receptor mechanism, or the organ of Corti. The phenomenon is generally absent in other forms of perceptive deafness, unless there has been an interference with the blood supply of the cochlea.

5. The presence of the recruitment phenomenon should, in the present state of knowledge, preclude the possibility of restorative hearing surgery on the inner ear.

6. The recruitment phenomenon indicates a gross lesion in the end-organ of hearing, with which there is generally speaking a decrease in speech intelligibility. The presence or absence of the loudness recruitment phenomenon, therefore, should be of some value in determining the probable usefulness of a hearing aid.

7. Persons with conductive deafness due to alterations in the middle ear mechanism do not exhibit the loudness recruitment phenomenon.

8. The absence of the loudness recruitment phenomenon in the presence of a perceptive deafness is generally indicative of a retrocochlear localization of the lesion. The topographic diagnostic significance of the loudness recruitment phenomenon should not be overestimated, and other investigations should not be neglected; however, the loudness recruitment phenomenon and the difference limen test appear, from anatomical, pathological, physiological and clinical points of view, to be valid and reliable. They have given the profession a means of differentiating cochlear from retrocochlear lesions in perceptive hearing losses, whether due to injury, infection or degenerations, and, as such, have added infinitely to our neuro-otological armamentarium.

REFERENCES.

1. Denes, P., and Naunton, R. F.: *Jour. Laryngol. and Otol.*, 64:375, 1950.

2. Dix, M. R., and Hood, J. D.: *Ibid.*, 67:343, 1953.

3. Dix, M. R.; Hallpike, C. S., and Hood, J. D.: *Proc. Roy. Soc. Med.*, 41:516, 1948.

4. Idem.: *Ibid.*, 42:685, 1949.

5. Dix, M. R.: *Brit. Med. Bull.*, 12:119, 1956.

6. Fowler, E. P.: *Arch. Otolaryngol.*, 24:731, 1936.

7. Hirsh, I. J.; Palva, T., and Goodman: *Ibid.*, 60:525, 1954.

8. Hood, J. D.: *Brit. Med. Bull.*, 12:125, 1956.

9. Huizing, H. C., and Reyntjes, J. A.: The Laryngoscope, 62:521, 1952.

10. Jerger, J. F.: *Ibid.*, 62:1316, 1952.

11. Idem.: *Jour. Speech and Hearing Disorders*, 20:1955.

12. Lorente de No, R.: *Trans. Amer. Otol. Soc.*, Vol. 27, 1937.

13. Idem.: The Laryngoscope, 43:1, 1933.

14. Idem.: *Ibid.*, 43:327, 1933.

15. Idem.: *Ibid.*, 47:373, 1937.

16. Lundborg, T.: *Acta Oto-Laryngol.*, Supp. 99, 1952.

17. Lurie, M.: *Ann. Otol., Rhinol. and Laryngol.*, 45:339, 1936.

18. Idem.: The Laryngoscope, 54:375, 1944.

....19. Lüscher, E.: *Proc. Roy. Soc. Med.*, 43:1116, 1950.

20. Palva, T.: The Laryngoscope, 65:829, 1955.

21. Schuknecht, H. C., and Woellner, R. C.: *Jour. Laryngol. and Otol.*, 69:75, 1955.

22. Schuknecht, H. C.: The Laryngoscope, 65:402, 1955.

23. Stevens, S. S., and Davis, H.: "Hearing." John Wiley & Sons, Inc., New York, 1937.

24. Wever, E. B.: "Theory of Hearing." John Wiley & Sons, Inc., New York, 1949.

25. Williams, H.: "Ménière's Disease." Charles C. Thomas, Springfield, Ill., 1952.

206 Medical Arts Bldg.

PENICILLIN RESISTANT GROUP-A STREPTOCOCCI IN NOSE AND THROAT INFECTIONS.

MERRILL LINEBACK, M.D.,

College Park, Ga.

In a recent discussion of antibiotic prophylaxis the statement was made: "... that thus far penicillin resistant strains of Group-A hemolytic streptococci . . . have not been encountered."[1] This is with reference to the human equation. An indication that all is not well with penicillin from the experimental standpoint is to be found in an article by Eagle,[2] who suggests that a delay in its use for treatment, or a refractory physiological state of the host, may be the reason for the paradoxical treatment failures with penicillin in Beta-hemolytic streptococcal infections in mice.

The purpose of this report is to present personal experiences with 22 cases of Group-A streptococcal infections seen in adults and gleaned over the past five years, which were found to be resistant to penicillin; 15 completely so, and seven of varying degrees of sensitivity. The Beta-hemolytic streptococcus was isolated in 17 cases, and the Gamma streptococcus or viridans in five (see Charts). All bacteriological work was done in the laboratory of the Georgia Baptist Hospital, Atlanta, Ga., and in each case grouping and typing according to the method of Lancefield was performed. The Beta-hemolytic streptococci were all Group-A, Type 4, and all were producers of hyaluronidase. Sensitivity testing was done both with the disc and serial dilution *in vitro* techniques. Interpretation of these studies in terms of living response and reaction may be fraught with hazard, but in general clinical effectiveness correlates well with the sensitivity tests.

The dangers of mishandling antibiotic therapy are still not realized by the majority of practitioners, as an increasing volume of literature on reactions, and even death, witnesses. The blithe assumption that: "There is no evidence that

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication May 15, 1958.

penicillin resistance is of much importance in the treatment of Beta-hemolytic streptococcal or certain other infections,"[3] is no longer tenable. Other authors[4] note: "No instances of development of resistance by Group-A hemolytic streptococci have been reported in the literature, nor did we encounter any when using monthly courses over a period of five years."

CHART I.

Lancefield Group-A	Tetracycline	Chloramphenicol	Erythromycin	Streptomycin	Dihydro-streptomycin	Oxytetracycline
Beta-hemolytic streptococcus (17 cases)	8	3	3	1	1	1
Streptococcus viridans (5 cases)	4	—	1	—	—	—

CHART II.

Resistance to Penicillin *in vitro* Testing Using 0.5 Units as Standard.

Lancefield Group-A	2X	3X	4X	5X
7 Cases	2	3	1	1

The common practice of giving penicillin "shots" for a "cold" is mentioned here only to be condemned.

Mangiarracine,[5] in an excellent article, emphasized the importance, in fact necessity, of accurate bacteriological work with sensitivity testing before the use of any antibiotic. Woodward[6] states that: "Before blindly administering an antibiotic, the physician should weigh all eventualities and be prepared to answer several questions:

1. Is an antibiotic really needed?

2. What is the proper drug and how long should it be continued?

3. Is the administration going to affect in any way the ultimate immune status of the patient?

4. Is the microbial agent sensitive to the antibiotic being employed, and does it readily acquire resistance to the drug?

5. Is dual therapy needed or can it be harmful?"

DISCUSSION.

Each of the 22 patients had previously received penicillin either orally or by the intra-muscular route; some had received it on occasion two or three years in succession for throat or nasal infection. Each was a treatment failure, in that for two or three weeks prior to examination and study they had received from their local physician two or more "shots" of penicillin without effect. The complaints ranged from a constant, unabating sore throat or nasopharyngitis, usually attended with mild fever and temperature elevation, to obvious prostration, dyspnea, dyshagia and odynphagia. Only the most unusual case of the series will be given in detail here. This one was resolved easily by a combination of precise bacteriological technique and serendipity.

CASE REPORT.

Mrs. H., a 33-year-old housewife, had received from her physician in the two years prior to this study a series of penicillin injections for a severe sore throat, associated with marked regional lymphadenopathy. Each time her disease had rapidly abated. No bacteriologic studies had been done. Two days prior to study she had received a "shot" of penicillin which had no effect on her throat. Examination of oropharynx revealed a grayish white mucous membrane and a normal larynx. Nasopharynx was unobstructed. A large thumb-sized mass was obvious in the left side of her neck just anterior to the sternomastoid muscle. This gland was slightly tender and very firm to the touch. She admitted that the node was less tender after the penicillin injection, but she now complained of dysphagia and inability to swallow soft foods. Water and other liquids were all she could handle. Temperature by mouth was 100 degrees F.

Because of her apprehension about the difficulty in swallowing she was admitted for observation and a culture was taken. During the night she experienced an episode of dyspnea and cyanosis of short interval. The next morning laryngeal examination revealed a marked redness to the oropharynx and a pearly whiteness to the larynx, which was obscured by a round ping-pong ball sized mass apparently arising from the left arytenoid. A quick check with the laboratory revealed that the previous day's culture was negative. The throat was recultured immediately and planted direct into media at the bedside. An emergency tracheotomy was considered, but a trial of ACTH and Cortisone was attempted. A Levin tube was inserted for feeding, she being unable to swallow liquids without severe pain. Since penicillin had proved ineffective and not knowing which antibiotic would prevail, certainly nothing by mouth or tube, it was decided to give her 2 grams of dihydro-

streptomycin I.M. empirically. In four hours dysphagia was less and she was more comfortable. No tracheotomy was needed.

Eighteen hours after the second culture was planted the growth was found to be Beta-hemolytic streptococcus, and the *only* antibiotic which was effective at all was dihydrostreptomycin. She was continued on this drug for three days, as well as on Cortisone for the laryngeal edema. Local heat in the form of the large wet-pack hydrocollator to the left side of the neck was used in addition, and in four days she was free of discomfort. The mass in the neck was pea-sized and the larynx had returned to its normal configuration at the time of discharge from the hospital.

In each instance the only comment that can be made is that prior to bacteriologic studies the patient had been in discomfort so that the normal performance of job, or housework, was impaired, and that at the conclusion of specific antibiotic therapy each had returned to a state of what to them was considered normal health and no discomfort was present.

A distinction should be made between the development of resistance by the streptococcus during the course of treatment with penicillin, and the infestation of the human economy with an organism that was resistant to penicillin from the outset.

In the opinion of the writer it is the latter aspect of the penicillin problem that is more important. It behooves the physician first to realize that such a state of affairs exists; second, since penicillin reactions are being reported more and more frequently, that he should culture and have sensitivity tests beforehand, and only *then* give the most effective antimicrobial agent as shown both by the disc and *in vitro* serial dilution techniques; third, that penicillin is the most common offender in causing thrush or moniliasis of the mouth and tongue, and therefore, should be used with caution, "covering" it with large doses of B-complex and mycostatin tablets, or suspension, or both concurrently. Gentian violet may often have to be used to control this complication. In the author's routine daily office practice oral moniliasis is seen in over 50 per cent of patients. This varies from groups of white patches on the buccal mucous membrane and palates, to the yellowish brown to black hairy tongue. In most instances each patient had received from another physician one or more "shots" of penicillin only to be made worse, with specific reference to the throat.

SUMMARY.

In view of the fact that it is a commonly held opinion that the Group-A streptococci are almost always responsive to penicillin therapy, these 22 cases in which penicillin was of no value at all should make the practitioner "fall back and regroup" his previous conceptions regarding its use, particularly in respiratory infections.

The Beta-hemolytic streptococcus was encountered in 17 cases. Tetracycline was the drug of choice in eight, chloramphenicol and erythromycin each three, streptomycin, dihydrostreptomycin, and oxytetracycline one each. The streptococcus viridans was found in five cases, tetracycline was the choice drug in four, erythromycin in one. There were seven cases of Group-A streptococcal infections in which penicillin was weakly effective, *i.e.*, it required two, three, four or five times the normal concentration to kill the offending organism.

It cannot be emphasized often enough that, in the management of any infection, before administering an antimicrobial agent, a smear from the involved region, culture and sensitivity testing should be done, and then only should the most effective drug be used.

Before giving any drug the practitioner should review the five precepts of Woodward outlined above.

REFERENCES.

1. Editorial: Antibiotic Prophylaxis. *New Eng. Jour. Med.*, 257:1084, Nov. 21, 1957.

2. Eagle, H.: Experimental Approach to the Problem of Treatment Failure with Penicillin: Group-A Streptococcal Infection in Mice. *Amer. Jour. Med.*, 13:389-399, Oct., 1952.

3. Long, P. H.: Mode of Action of Antibiotics (A. Walter Suiter Lecture). *N. Y. St. Jour. Med.*, 52:1637-1639, July 1, 1952.

4. Kohn, K. H.; Milzer, A., and MacLean, H.: Prophylaxis of Recurrence of Rheumatic Fever with Penicillin Given Orally; Final Report of Five-Year Study. *J.A.M.A.*, 151:347-351, Jan. 31, 1953.

5. Mangiarracine, A. B.: Modern Bacteriology as an Aid to the Otolaryngologist. *Ann. Otol., Rhinol. and Laryngol.*, 60:230, 1951.

6. Woodward, T. E.: "The Chloramphenicol Problem," Special Article in *Mod. Med.*, p. 68, Jan. 1, 1953.

124 West Princeton Ave.

SOME HISTORICAL NOTES ABOUT THE FIRST LARYNGECTOMIES.*

ADOLF W. SCHWARTZ, M.D.,
and
KENNETH D. DEVINE, M.D.,

Rochester, Minn.

Long before the discovery of blood groups, without knowledge about electrolytes, without the intravenous use of fluids, and before antibiotics and other modern drugs, great surgeons performed operations which still belong among the most difficult, as well as major operations. Almost a century has passed since the first successful laryngectomy was performed, and today, 85 years after performance of the first recorded total laryngectomy on a patient by Dr. Billroth,[1] a review of his results, technique, and of some of the experience of this father pioneer surgeon should be of some historical interest.

Before this operation took place, Dr. Czerny[2] had performed experimental laryngectomies on dogs; he also had constructed a T-shaped tube which he placed in the wound to serve as an "artificial voice box." In 1829 Dr. Albers[3] recorded the successful extirpation of the larynx of a dog which lived for nine days. Drs. von Langenbeck[3] (1854), Köberle[3] (1856) and Hueter[3] discussed and proposed the laryngectomy, but it was not before December 31, 1873, that Dr. Billroth performed the first laryngectomy on a human being. The detailed description was presented by Dr. Billroth's assistant, Dr. C. Gussenbauer,[1] to the Third Congress of the German Surgical Society on April 11, 1874. It is interesting to find also that on the day before this presentation to the Third Congress, Dr. Küster[4] presented to the same congress "a newer method of blood transfusion from animals to humans,

*The Mayo Foundation, Rochester, Minn., is a part of the Graduate School of the University of Minnesota.

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication October 10, 1958.

using the arterial blood pressure from muttons," which illustrates medical progress in another field since that time.

The entire procedure of total laryngectomy took Dr. Billroth one hour and 45 minutes.

The patient, a 36-year-old teacher with a tumor below the true vocal cords, was first treated with multiple cauterizations and biopsies (microscopic report: epithelial carcinoma of the larynx). On November 21, 1873 (after a preliminary tracheotomy), Dr. Billroth performed a laryngofissure and removed the tumor with scissors and sharp curette. The main tumor was situated on the left side, so the surgeon was able to preserve the right vocal cord; the wound edges were approximated with tape. On the second postoperative day the patient began to have septic temperatures, and infection developed in the wound, which was treated with a preparation containing silver nitrate; on the seventh postoperative day the patient was up and about, and two days later a high degree of dyspnea developed. The wound was reopened and examination revealed that the growth had recurred.

In an attempt to curette the larynx, Dr. Billroth reopened the wound, but discovered that the malignant growth had invaded the cartilage. The patient was awakened, informed of the necessity to remove the larynx and again anesthetized. The technique consisted mainly in dull dissection of the larynx on both sides while the assistant exerted a pull on the larynx. The trachea was divided just below the cricoid, the larynx was pulled forward and up, and dissected from the anterior wall of the esophagus; the thyrohyoid membrane was divided last. The anesthesia was not sufficient, and the operative procedure was frequently interrupted by strong coughing spells during which blood in rather large amounts was expelled from the trachea. The bleeding was controlled by pressure with large sponges.

The growth must have been quite large since, after examining the specimen, Dr. Billroth decided to remove most of the epiglottis and the first two tracheal rings. The tracheal stoma was fixed to the skin with one suture on each side and the opening into the pharynx was made smaller by applying three sutures. Four hours later, during a bout of coughing, a profuse hemorrhage developed from the left superior laryngeal artery which required a ligature. Dr. Gussenbauer[1] remarked about the large amounts of coagulated blood which the patient had coughed up without having had great signs of dyspnea. Three days after the operation the sutures and the ligatures were removed. The wound represented a groove with the tracheal stoma in the lower end and the opening into the pharynx on the upper end.

Fortunately the patient escaped all secondary complications, such as shock, aspiration pneumonia, mediastinitis, sepsis, meningitis, tetanus and large abscess. The pharyngeal fistula began to close early, and the patient was started on a soft diet on the eighth postoperative day; the gastric feeding tube was removed on the eighteenth day. The patient was dismissed on March 3, 1874, after he had learned how to use the artificial larynx designed by Dr. Gussenbauer (see Fig. 1). Unfortunately, a recurrence of the lesion with metastatic nodes developed, and the patient died approximately one year after the operation.

The second recorded total laryngectomy for carcinoma of the larynx was performed in April, 1874, by Dr. Heine[3] of

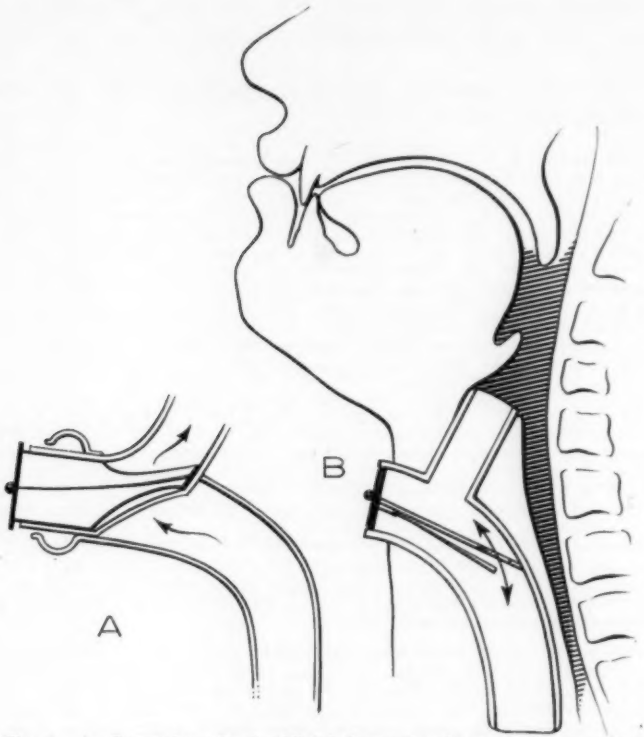

Fig. 1. A—Gussenbauer's "artificial larynx" with a metal membrane. B—A modification by Foulis. (Adopted from M. Schüller, 1880.)

Prague. The patient survived for six months; in June of the same year Dr. Maas[5] of Breslau performed the third laryngectomy. Pneumonia developed, and the patient died two weeks after operation. Dr. Schmidt[6] of Frankfurt performed the fourth laryngectomy on August 12, 1874, but the 56-year-old patient survived only four days and died of collapse. Dr. Billroth performed his second total laryngectomy combined with removal of a small goiter on November 11, 1874, on a 54-year-old man. The patient suffered preoperatively of bronchitis and died of bronchopneumonia on the fourth day after operation.

The first successful laryngectomy with many years of survival was performed on a 24-year-old man in 1875 in Italy by Dr. Bottini.[3] The lesion was a mixed round and spindle cell sarcoma. The patient lost a large amount of blood and erysipelas developed; but he survived, and for years he was able to continue his work as a mail carrier in a mountainous region.

Notable advance in the technique of total laryngectomy was reported by Dr. von Langenbeck.[7] On July 28, 1875, he presented an unusual specimen during the meeting of the Medical Society in Berlin. It included the entire larynx with epiglottis, hyoid, a third of the tongue, the anterior and lateral portion of the pharynx, a portion of the esophagus, both submaxillary glands and lymphatic tissue from the submaxillary triangle. A 57-year-old man with carcinoma of the larynx had undergone tracheotomy but refused to have a laryngectomy. He returned seven months after the operation, and on July 21 was operated on by Dr. von Langenbeck. The incision was T-shaped. The dissection was performed from above, so that the trachea was severed last. The time involved was a little more than two hours. It is recorded that the operation was done as exactly as an anatomic dissection, and in spite of the extensive region involved, the blood loss was unusually small, because each vessel was ligated prior to being cut. Forty-one ligatures were made, including both external carotid arteries, lingual, facial, superior thyroids, etc. The patient survived for four months but died on November 23, 1875, of collapse during an attempt to remove the metastatically involved glands of the left side of the neck. This operation of Dr. von Langenbeck could be considered the forerunner of the combined wide-field laryngectomy and neck dissection.

It would be too much to list each laryngectomy in the following years, but mention of one is worth while. Again it was Dr. Billroth who, in July, 1878, performed the surgical removal of half of the larynx. He was probably the first to do a hemilaryngectomy, or partial laryngectomy. In this patient only the left side of the larynx was removed.

TECHNIQUE.

In the first ten years after Dr. Billroth's successful laryngectomy, the advantage of a preliminary tracheotomy (performed, if possible within a few weeks to months before the main operation) was soon recognized. The Trendelenburg position, the Trendelenburg cuffed tracheotomy tube (see

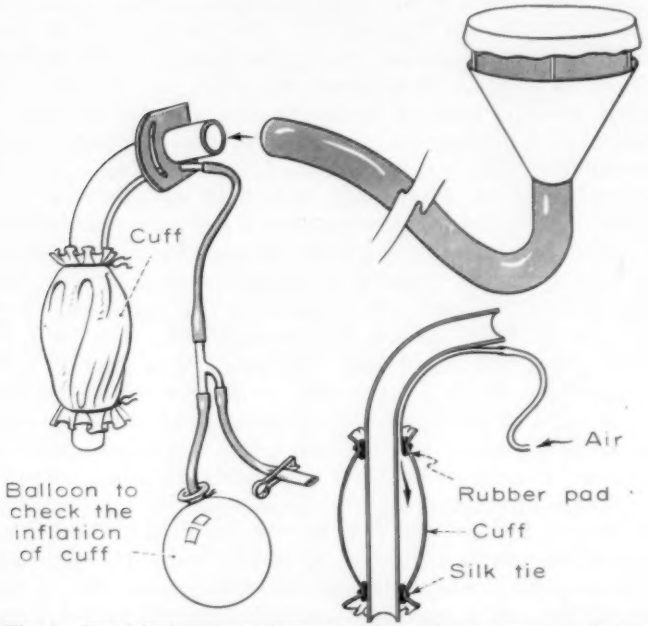

Fig. 2. Trendelenburg's "tampon-cannula" used for oral operations and operations on the upper respiratory tract as early as 1865 to 1870. Chloroform was sprayed on gauze over funnel. (Modified after M. Schüller, 1880.)

Fig. 2), and the use of cautery on bleeding points were soon recognized as great technical aids. The incisions in the first three or four operations were mid-longitudinal, but Dr. von Langenbeck extended his incision laterally to the medial border of each sternocleido muscle and produced a T-shaped incision. After penetration through the muscle layer in the

midline, the larynx was isolated mainly by blunt dissection aided by a strong pull on the larynx anteriorly, or to the opposite side of the dissection. The separation was started mostly from above and carried through the plane of separation from the anterior esophageal wall; the trachea was then cut posteriorly. This is not exactly the technique used by Dr. Billroth, and many modifications soon developed.

The principle of the postoperative treatment in those early days was to leave the wound wide open. The wound was packed with sponges soaked in carbol solution to soak up the saliva and the wound secretions. The outer dressing produced counter pressure to eliminate dead space. The trachea was anchored to the skin with a few silk sutures, and the tracheal cannula was inserted. The dressings were changed daily, and the wound was irrigated with carbol solution.

The oral cavity was washed out several times daily with disinfectant solutions, and the patient was fed by a gastric tube. A steam kettle evaporating turpentine oil with carbolic acid and glycerine was placed in the room. In those days it was a common procedure to introduce an artificial larynx into the wound in the early postoperative period.

ARTIFICIAL LARYNGES.

The early artificial larynges were of various design, but most of them incorporated a vibrating membrane in the course of a tube connecting the tracheal stoma with the pharyngeal stoma. The membranes were of ivory, horn, silver, copper, or hard rubber, but all had the disadvantage of soon being covered with thick mucus and all presented an obstacle to breathing and talking. Saliva and food particles easily entered the trachea producing violent coughing spells. Dr. Billroth placed the artificial larynx in his first patient on the twenty-first day after operation; Dr. Maas placed his on the eleventh day, Drs. Heine and Czerny on the ninth and Dr. Schmidt[6] immediately after removal of the larynx. Generally, however, a period of two weeks was adopted as

the most suitable time, and soon it was made evident that speech was possible without an artificial larynx.

The purpose of this article is to describe the first steps taken toward the well-developed technique of total laryngectomy as used today. In the early days, total laryngectomy was considered to be an exceedingly dangerous operation. Between 1881 and 1888, Dr. Sendziek[8] collected data of 110 total laryngectomies, and between 1888 and 1894 only 47. It should be mentioned briefly that with the publication of Dr. Röntgen's communication on a new kind of rays[9] in 1898, and with the isolation of radioactive substance by his assistant Dr. Maria Sklodowska (later Madame Curie), the surgical treatment of malignant involvement of the larynx was soon more and more frequently replaced by radiation therapy. The higher rate of complications and the high mortality rate due to surgical treatment strengthened the hand of the radiologist and reversed the trend toward increasing surgical intervention in carcinoma of the larynx.

Among the few who stood out in favor of surgical intervention were Drs. Jackson,[10] Lewis,[10] Thomson,[11] New[12] and others. The general surgeons were conspicuous by their absence, and it was not until the advancement in medicine of the last 20 years that total wide-field laryngectomy became widely accepted as a standard procedure in the treatment for cancer of the larynx.

REFERENCES.

1. Gussenbauer, Carl: Ueber die Erste durch Th. Billroth am Menschen Ausgeführte Kehlkopf-Exstirpation und die Anwendung eines Künstlichen Kehlkopfes. *Arch. Klin. Chir.*, 17:343-356, 1874.

2. Czerny, V.: Versuche über Kehlkopfexstirpation. Wien. Med. Wchnschr., 20:557-561, May 14, 1870.

3. Schüller, Max: "Die Tracheotomie, Laryngotomie und Exstirpation des Kehlkopfes." In: Deutsche Chirurgie, Vol. 37:228. F. Enke, Stuttgart, 1880.

4. Küster, Ernst: Ueber die directe Arterielle Thierblut-Transfusion. *Arch. Klin. Chir.*, 17:385-404, 1874.

5. Maas, H.: Vollständige Exstirpation des Kehlkopfes; Tod nach 14 Tagen. *Arch. Klin. Chir.*, 19:507-513, 1876.

6. Schmidt, Moritz: Totalexstirpation des Kehlkopfes mit ungünstigem Ausgange. *Arch. Klin. Chir.*, 18:189-194, 1875.

7. von Langenbeck, B.: Total Extirpation des Kehlkopfs mit dem

Zungenbein; einem Theil der Zunge, des Pharynx und Oesophagus. *Berl. Klin. Wchnschr.*, 12:453-455, Aug. 16, 1875.

8. BUTLIN, H. T.: "On the Operative Surgery of Malignant Disease." Ed. 2:426. P. Blakiston's Son & Co., Philadelphia, 1900.

9. RÖNTGEN, W. C.: 1. Ueber eine neue Art von Strahlen; 2. Ueber eine neue Art von Strahlen; 3. Weitere Beobachtungen über die Eigenschaften der X-Strahlen. *Ann. d. Phys. u. Chem.*, 64:1-37, 1898.

10. LEWIS, F. O.: Laryngeal Cancer, Its Treatment, with Special Reference to Total Laryngectomy. THE LARYNGOSCOPE, 35:607-610, Aug., 1925.

11. THOMSON, ST. CLAIR: "Diseases of the Nose and Throat; Comprising Affections of the Trachea and Oesophagus." Ed. 3:791. Cassell & Company, Ltd., New York, 1911.

12. NEW, G. B.: Surgical Diathermy in Laryngology. *Arch. Otolaryngol.*, 3:301-304, April, 1926.

POST GRADUATE COURSES AT TEMPLE UNIVERSITY SCHOOL OF MEDICINE AND HOSPITAL.

A Postgraduate Course in Bronchoesophagology, February 16th to 27th, 1959, and a Postgraduate Course in Laryngology and Laryngeal Surgery, April 13th to 24th, 1959, will be given by Temple University School of Medicine.

These courses are to be given in the Department of Laryngology and Broncho-Esophagology, Temple University Medical Center, under the direction of Drs. Chevalier L. Jackson and Charles M. Norris.

The tuition fee for each course is $250.00. Application blanks and further information concerning dates of subsequent courses may be obtained by writing to: Jackson-Research, Laboratory 604, Temple University Medical School, 3400 N. Broad Street, Philadelphia 40, Pa.

ORIGIN OF OTORHINOLARYNGOLOGIC TERMS.*

GENE V. WILLIAMS, M.D.,

Kansas City, Kans.

The physician is frequently confronted with the question, "Why do doctors use such big words and what do they mean?". The reaction of the doctor to this usually abrupt and often petulant query may vary from arrogant intellectual impatience to shoe-scuffing self-modesty. Occasionally he finds himself hopelessly cornered and is forced to admit honestly, at least to himself, that he just does not know what a particular word means. As a case in point he undoubtedly knows what the thyroid gland is and what it does; he could quite probably describe the organ ad nauseum. What he may or may not know is that the word thyroid is derived from a Greek word which applied to a particular type of shield. This long shield which had a deep notch at the top for the chin was carried exclusively by the Minoan foot soldiers of ancient Crete, and its singular shape suggested to Galen the name for the cartilage in the neck. The gland takes its name in turn from that of the cartilage (see Fig. 1).

Upon hearing such a tale as this the busy clinician will probably counter with something to the effect that he knows perfectly well the anatomy and physiology of the structure in question. He may further declare that it is not only unnecessary for him to clutter his mind with such trivia, but also that he just plain doesn't give a hoot what word the Greeks had for it. One must admit he has a point; however, the true meaning and derivation of the words commonly employed in medical circles can be very interesting.

These words are often descriptive, thus facilitating the conjuration of a mental image of the anatomical part. They may be of historical interest or simply tickle one's sense of

*From the Department of Otorhinolaryngology of the University of Kansas School of Medicine.

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication Sept. 30, 1958.

appreciation for things whimsical by their very quaintness. One could appeal to the nobler instincts by a lengthy discussion of how such a study might prove intellectually broadening, stimulate new interests, fill cultural gaps and help burnish off any remaining ragged burs of knowledge concerning one's chosen field of study. One could choose the easy road by simply calling it a hobby and thinking of it as fun.

Words, both conversational and scientific, take their origin

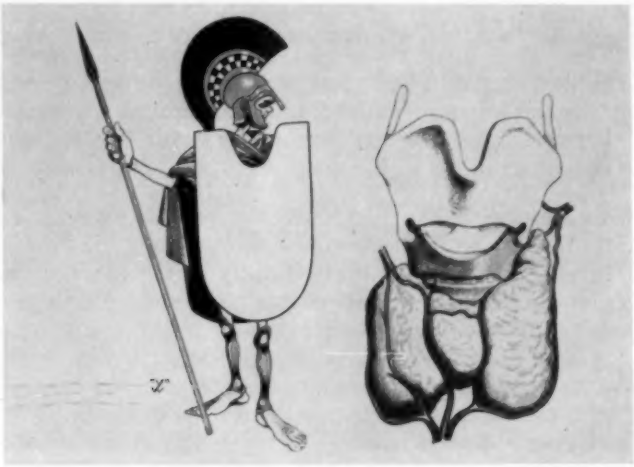

Fig. 1.

from many sources. The exact source may be unknown or at best controversial. Some come down to us, more or less intact, from the mouths of the ancients. There are those which are misnomers and others which have gradually evolved by being passed from one racial or cultural stock to another; while many of our words were, and still are being, "coined" by present day scholars.

Sanskrit, the ancient Aryan language of the Hindus of India, is of special interest to students of philology. It retains many of the characteristics of the parent Indo-European

language. Antrum, the Latin word for cave or cavity, is from a Greek word which was in turn derived from an earlier Sanskrit word meaning "within." The maxillary and frontal sinuses were noted and recorded by Leonardo da Vinci. Casserius, during the latter half of the Sixteenth Century, referred to the "Antrum Maxillae." The maxillary sinus was later called the "Antrum of Highmore" after the Seventeenth Century English naturalist. Genu, the Latin word for knee, is related to the Greek "gony." Both words are probably derived from the Sanskrit word "Janu." The geniculate body, being located on the facial nerve where it makes a "knee-like" bend in the facial canal thus becomes a more meaningful term.

The word "nose" is of uncertain origin, but arises from a root common to many languages. The Sanskrit word, nasa; the Latin, nasus; the Anglo Saxon, nosu; the German, nase; the Italian, naso; and the French nez are but a few examples.

The ancient Greeks had many words for many things. Carotid is derived from the Greek word meaning to "throttle or stupefy." During the First Century of the Christian era, Rufus of Ephesus referred to the ancients' awareness of "sleep" resulting from pressure being applied to the vessels of the neck. Referring to a blood vessel as an "Artery" stems from the ancient notion that these vessels contain air. The blame for this misconception is usually laid at the feet of Praxagoras, but one should not be too harsh in his judgment of the poor fellow, because Erasistratus agreed with him. It seems that Herophilus knew the vessels contained blood, but even with Galen supporting him the idea of air in the arteries persisted for a long time.

Since, to the ancients' minds, the windpipe was "also" an air-containing tube, it was called the "Arteria Trachea," or rough artery. Very early in the Sixteenth Century, the word "Arteria" was dropped, and the windpipe was referred to simply as the trachea. Making an opening in the trachea was first advocated by the followers of Aesculapius, and may have been performed by Paul of Aegina and Antyllus; however, the first recorded instance of a successful performance of this operation was by Brasovolus during the first half of

the Sixteenth Century. The procedure was called a "Bronchotomy" until 1718 when Heister introduced the term "Tracheotomy."

The word bronchus is said to be derived from the Greek word meaning to "pour or moisten." This is the result of an ancient belief that solid foods were carried to the stomach via the esophagus and liquids by way of the bronchi.

Parotid simply means "beside the ear" and refers to the parotid gland. The function of this gland was first demonstrated by Stensen in 1662.

Isthmus, though Latin, is from the Greek word meaning a narrow passage or opening, and also refers to a neck of land separating two bodies of water.

The earliest source of the word "Pharynx" is uncertain, but opinion allies it to the Greek word meaning "Gully or ravine." Galen defined the pharynx as the common opening of the gullet and windpipe. The word first appeared in English medical terminology in 1693.

The derivation of "Palate" is also questionable. It may be associated with pascere, "to feed," or, because the palate is surrounded by teeth, to palere, "to hedge in." Skeets says the root is uncertain, but Campbell believes it refers to Palatine Hill in Rome. In his opinion, the hill takes its name from balatans, "bleating," because of the bleating flocks that grazed there in antiquity. Most authorities feel that it is much less complicated and that Palatine is derived from palatium, "a palace." Before Fallopius differentiated between the hard and soft palates, they were jointly referred to as the "diaphragm oris."

Helix means a spiral, twisted, or coiled structure, and was used by Theophrastus some twenty-three Centuries ago in referring to the tendrils of vines. Aristotle employed the word when describing convolutions of the snailshell and loops of intestines (see Fig. 2).

The hyoid, "U-shaped," bone was first named and described by Herophilus, a Greek physician born in Alexandria in 344 B.C. (see Fig. 3).

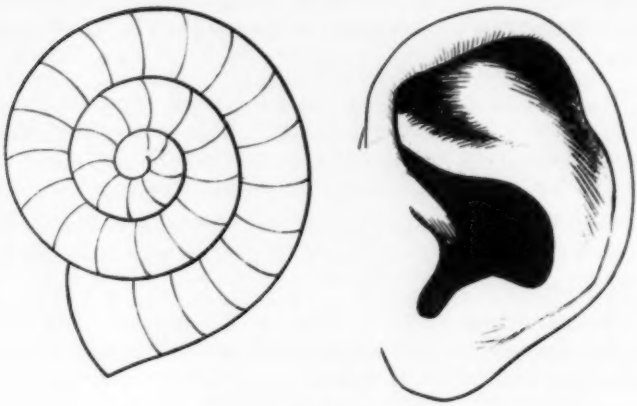

Fig. 2.

Fig. 3.

One Julius Pollux, an obscure Greek author who lived from 134 to 192 A.D., bequeathed his "Onomasticon," or "Vocabulary" to posterity. One section of this work concerns itself with medical terminology. The vocabulary was reprinted in 1502 and became an important source of words for those translators who busied themselves by replacing Arabic with Greek and Latin words. Such words as cricoid (see Fig. 4), meaning "ring-shaped" and tragus, meaning "goat," so named because of the hairy nature of the skin in this area, were introduced by Julius Pollux.

We have words which are the result of "slips of the pen;" for example, the word lacrimal is a Latin word meaning "tear." It is probably a misnomer due to an ancient scribe's error in copying "lakry" for "dakvy," the Greek word for tear. In support of this "careless scribe theory" some scholars state that there is an older form of the Latin word spelled d-a-c-r-i-m-a. The term "lacrimalis," first appeared in the English language some time before 1400.

There exists a rather lengthy list of words, commonly employed in the practice of medicine, which one might refer

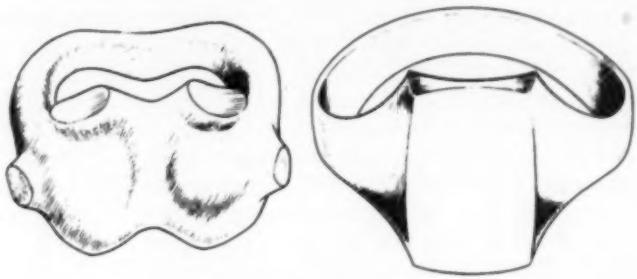

Fig. 4.

to as "Galenisms." Claudius Galen, often called the "Prince of Physicians," though more often referred to as just plain "Galen," was born in 130 A.D. at Pergamos. His father, Nikon, was an Asiatic Greek, and it is said that little Claudius inherited his mother's irritable temper. Galen removed to and settled in Rome when 30 years of age, and he became a famous physician as well as a prolific writer. His words were concerned, for the most part, with his own observations and experimentations, but he also found time to write a summary of the anatomical knowledge of his day. He died a septuagenarian in Sicily but remained influential from the grave. For 1,400 years he continued as the leading medical authority of the Christian world. Such anatomical structures as the ethmoid, "sieve-like," and sphenoid, "wedge-shaped," bones, as well as the pterygoid, "wing-like," and mastoid,

"breast-like," processes were first described and named by Galen. He observed that, in certain animals, the appearance formed by two small cartilages in the larynx, when covered by mucous membrane, was not unlike that of the mouth of a pitcher; therefore, the term arytenoid, meaning "pitcher-like," was applied to the cartilages. Galen first used the word glottis, the Attic form of the Greek word meaning "tongue," but also the name of the reed on a wood-wind instrument, as in the modern sense, meaning the mouth of the windpipe.

Hot on the heels of the Greeks tramped the Romans. Much of our medical vocabulary is Latin in origin. The etymology of malar, meaning "cheek," is uncertain. Association with malum, an apple, has been suggested. The term is also said to be a contraction of maxilla. Another word of debatable etymology is ala. It is suggested that it may be a contraction of axilla, as mala is perhaps a contraction of maxilla. It is interpreted as meaning "wing" or any side compartment. Our word tonsil is derived from the Latin tonsilla, meaning a "mooring stake" or any sharp-pointed pole stuck in the ground. Removal of the tonsils was described in the First Century by Celsus, who advocated enucleation with the finger. "Tonsillectomy" is a hybrid Greco-Roman term. Since the tonsil was once known as the "almond of the throat," pure Greek might be amygdalectomy or "removal of the almond." Sinus is Latin for anything hollowed out or curved, and is thus used for various unrelated structures. Mucus is a Latin word from the Greek word meaning "slime." Meatus, meaning a passage or channel, takes its origin from meare, "to go" or "to pass." From the Latin word tempus, meaning time, comes the English word temple. Both probably originate from the Greek word meaning "to cut." The temple was considered as a fatal and vulnerable spot whereupon a blow might well "cut off" one's life. The often-observed phenomenon of hair beginning to turn gray in this area emphasized the relationship of the area to fleeting tempus, or time. Jugular is from jugulum, the Latin word for throat. Jugulum is, in turn, derived from jugum, or yoke, because a yoke is carried on the neck.

There are many descriptive words in Latin. Lingua, which means tongue, is said to be imitative of the sound produced by licking with the tongue. Another descriptive Latin word is vomer, which means "ploughshare." The word is supposedly derived from vomere, "to vomit," because the plough "throws up" the newly broken soil to the side. The resemblance of the bone of the nasal septum to a plough-share is obvious (see Fig. 5). Piriform means "pear-shaped." Uvula, meaning "little grape," is a term which was not used by the

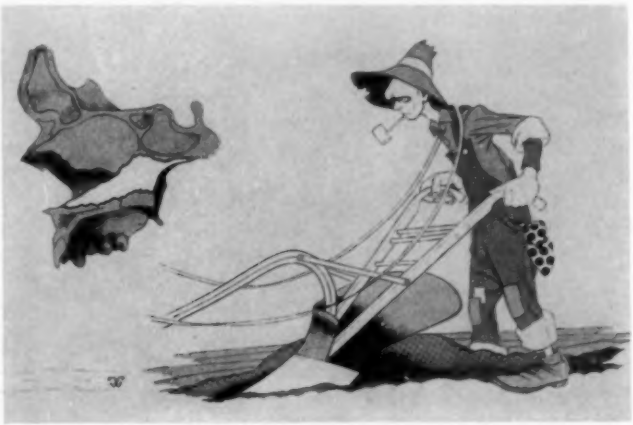

Fig. 5.

ancients. This dangling mid-line structure was called the "pillar" by the Greeks and "columella" by the Romans. Guy de Chauliac, a French surgeon, who practiced in Avignon during the first half of the Fourteenth Century, introduced the term "uvula." Frenulum is the diminutive of fraenum, meaning "bridle." Bulla means bubble or ball. Crus, which refers to the shin or leg, is applied to any leg-like structure (see Fig. 6).

The Anglo-Saxons have given us such words as ear, from eare; nose, from nosu; and nostril, from nosethyrl. Tongue is from an earlier Teutonic root, tonga or tunga.

The meanings of the Latin names for the three small ossicles found in the middle ear are familiar to all. Gabriello Fallopius accredited Berengarius of Capri, an Italian anatomist and surgeon, of the late Fifteenth and early Sixteenth Centuries, as the first to describe two of these small bones. Some authorities give this credit to Achillini of Bologna, an elder contemporary of Berengarius. Whoever may have been the first to observe and describe them, Vesalius, noted the

Fig. 6.

similarity in appearance of one of them to an anvil and the other to a hammer. He named them the incus and malleus respectively in 1543. In 1546, Ingrassias, an Italian anatomist, named the third stirrup-shaped ossicle the stapes. This third ossicle was not mentioned by Vesalius. The word stapes is known to be late Latin, because the Romans rode their horses "bare back." Stirrups suspended from the saddle did not appear on the scene until the Fourth Century (see Fig. 7).

Though Galen noted it and Vesalius described it, the name "labyrinth" was given to the coiled structure in the inner ear

by Fallopius in 1550. The name, of course, refers to the semi-mythical Cretan maze erected by Daedalus, within whose structure prowled the cannibalistic Minotaur, a monster half bull and half man, which was eventually destroyed by the hero Theseus.

Some structures have been given the names of those scholars who helped shed the light of knowledge upon them. The tubu-

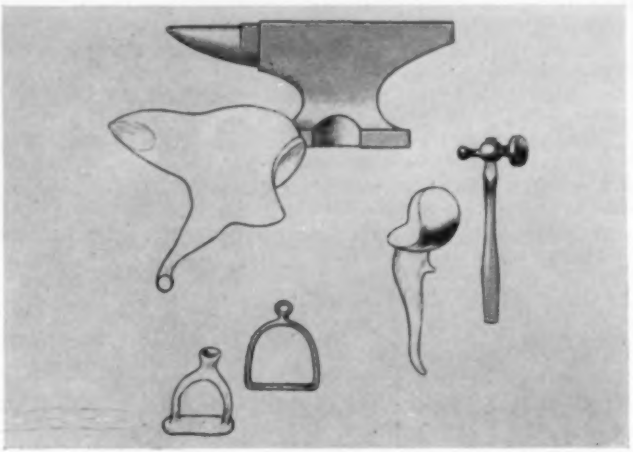

Fig. 7.

lar connection between the pharynx and middle ear was known to the ancients, having been dissected by Alcmaeon in 500 A.D. and described by Aristotle. The name Eustachian tube was given it by Val Salva in honor of Bartholommeo Eustochio, in 1704.

Hypertrophied lymphoid tissue in the nasopharynx was first described by Hubert Von Luschka during the latter half of the Nineteenth Century. The name adenoid, meaning "gland like," was not, however, new. Adenoides or corpus adenoides was an old term for the prostate gland.

In finding one's self so far afield from those regions which

are the province of the otorhinolaryngologist it is perhaps apropos that the discussion be concluded. This is not, by any means, submitted as an exhaustive etymological study. It is presented more as a stimulus to those who are susceptible, and as an appetizer for those whose intellectual palates have not been displeased.

BIBLIOGRAPHY.

1. "Blakiston's New Gould Medical Dictionary." Editors: Hoerr, Normand L., M.D., and Arthur Osol, Ph.D. The Blakiston Division, McGraw-Hill Book Company, Inc., New York, 1956.

2. "Composition of Scientific Words:" Brown, Roland Wilbur. Published by author, 1954.

3. "Dorland's Illustrated Medical Dictionary," 23rd Edition. W. B. Saunders Company, Philadelphia, 1957.

4. "Etymological Dictionary of the English Language:" Skeet, Walter W. Oxford University Press, London, 1956.

5. "MacMillan Medical Dictionary." Editor: Wakely, Sir Cecil. The MacMillan Company, New York, 1954.

6. The Origin of Medical Terms:" Skinner, Harry Alan. The Williams & Wilkins Company, Baltimore, 1949.

ALUMNI ASSOCIATION OF THE NEW YORK EYE AND EAR INFIRMARY.

The Annual Spring Meeting of the Alumni Association of the New York Eye and Ear Infirmary last April was so well received that it has been decided to expand next year's meeting, which will take place from April 20-23, 1959.

Symposia and lectures on Hearing Rehabilitation, Endoscopy, and Ear Surgery will be conducted. It is also planned to offer refresher courses in Mastoid and Fenestration Surgery and Stapes Mobilization Techniques.

More complete information regarding the meeting will appear in a later issue of THE LARYNGOSCOPE.

PROGRAM FOR THE SCIENTIFIC SESSION OF THE THE AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

March 10, 11, 12, 1959

"Neck Dissection: A Clinico-Anatomical Study of 200 Cases."
George F .Reed, M.D., Boston, Mass.

"Evaluation of Laryngectomy with Radical Neck Dissection."
John J. O'Keefe, M.D., Philadelphia, Pa.
Open Discussion.

"Progress in Parotid Surgery."
Robert E. Boswell, Dayton, O.

"Cystadenolymphomas of the Parotid Gland."
John F. Daly, M.D., New York City.
Open Discussion.

"Experience with Trans-Septal-Sphenoid Subtotal Hypophysectomy in Advanced Breast Cancer."
Walter E. Heck, M.D.; Robert C. McNaught, M.D.; Leonard G. Dobson, M.D., (by invitation); and Francis S. Greenspan, M.D., (by invitation), all of San Francisco, Calif.
Open Discussion.

"The Problem of Sinusitis in Children."
Victor R. Alfaro, M.D., Washington, D. C.
Open Discussion.

"Endocrine Aspects of Meniere's Disease."
George E. Shambaugh, Jr., M.D., Chicago, Ill.
Open Discussion.

"Cholesteatoma of the External Auditory Canal."
Lester A. Brown, M.D., Atlanta, Ga.
Open Discussion.

"The Diagnosis and Treatment of Facial Paralysis, Secondary to Basal Skull Fracture."
Miles L. Lewis, Jr., M.D., New Orleans, La.
Open Discussion.

"Temporary Obstruction of the Arterial Supply of the Labyrinth."
Henry Perlman, M.D., Chicago, Ill.
Open Discussion.

"Otolaryngologic Aspects of Cerebrovascular Disease."
Bruce Proctor, M.D., Detroit, Mich.
Open Discussion.

"Juvenile Nasopharyngeal Angiofibroma."
Maurice Schiff, M.D., Oakland, Calif.
Open Discussion.

"Late Hearing Results in Mobilization Surgery."
Samuel Rosen, M.D., New York City, and
Clair M. Kos, M.D., Iowa City, Ia.
Open Discussion.

"Trends in Mobilization Surgery."
Howard P. House, M.D., Los Angeles, Calif.

UNIVERSITY OF MICHIGAN MEDICAL CENTER.

The annual Otolaryngology Conference at the University of Michigan will be held April 16, 17 and 18, 1959, under the direction of Dr. James H. Maxwell, Chairman of the Department of Otolaryngology.

Applications may be addressed to the Department of Postgraduate Medicine, University Hospital, Ann Arbor, Mich.

PROGRAM OF THE NINETY-SECOND ANNUAL MEETING OF THE AMERICAN OTOLOGICAL SOCIETY.

Hot Springs, Va., March 13-14, 1959.

The Cochlear Potentials and Their Relation to Hearing.
E. Glen Wever, Ph.D.

More About the Physiology of the Cochlea.
Hallowell Davis, M.D.

Audiometry by Electroencephalography.
A. J. Derbyshire, M.D. (by invitation).

Ototoxicity of Kanamycin.
J. E. Hawkins, Jr., Ph.D. (by invitation).

A Cause of Sudden Deafness.
William Saunders, M.D. (by invitation).
William Libby, M.D. (by invitation).

Acoustic Trauma Following Intermittent Exposure to Tones and Noise.
Walter Covell, M.D. (by invitation).
Don Eldredge, M.D. (by invitation).

An Experimental Study of the Dynamic Circulation of the Labyrinthine Fluids of Living Mammals.
Francis L. Weille, M.D.;
John W. Irwin, M.D. (by invitation).
Coza Jako, M.D. (by invitation).
Patricia Palmer, A.B. (by invitation).

Long Term Results from Surgery for Otosclerosis.
Theodore E. Walsh, M.D.

Ultra-Sonic Therapy of Meniere's Disease.
Franz Altmann, M.D.

Presbycusis.
Edmund P. Fowler, M.D.

Nihil Sub Sole Novum.
Lyle Sellers, M.D.

Otic Embryopathy.
George Kelemen, M.D.

Cochlear Blood Flow in Hypothermia.
H. B. Perlman, M.D.;
Robert Butler, M.D., and
Robert Kimura (by invitation).

Postural Nystagmus Produced by Cerebellar Lesions in the Cat.
Cesar Fernandez, M.D. (by invitation).

Fluid Ears and Nerve Deafness, Circa 1835.
Gordon Hoople, M.D.
Wesley H. Bradley, M.D. (by invitation).

Tympanoplasty.
Fred Guilford, M.D.

Surgical Treatment of Facial Paralysis and Associated Conductive Deafness in Fracture of the Temporal Bone.
H. E. McHugh, M.D.

POSTGRADUATE MEDICAL INSTRUCTION DEPARTMENT—THE MOUNT SINAI HOSPITAL.

An intensive Course in Rhinoplasty, Reconstructive Surgery of the Nasal Septum and Otoplasty will be given July 11, to July 24, 1959, by Dr. Irving B. Goldman and staff at the Mount Sinai Hospital in affiliation with Columbia University.

Candidates for the Course should apply to Registrar for Postgraduate Medical Instruction, The Mount Sinai Hospital, Fifth Avenue and 100th Street, New York 29, New York.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

(Secretaries of the various societies are requested to keep this information up to date).

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Erling W. Hansen, 90 So. Ninth St., Minneapolis, Minn.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Meeting: Palmer House, Chicago, Ill., Oct. 10-15, 1959.

AMERICAN BOARD OF OTOLARYNGOLOGY.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION.

President: Dr. Francis W. Davison, Geisinger Memorial Hospital, Danville, Pa.
Vice-President: Dr. George S. McReynolds, 615 N. Boulevard, Galveston, Tex.
Secretary: Dr. F. Johnson Putney, 1712 Locust St., Philadelphia 3, Pa.
Treasurer: Dr. Verling K. Hart, 106 W. 7th St., Charlotte, N. C.
Meeting: The Homestead, Hot Springs, Va., March 10-11, 1959.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

President: Dr. Fred W. Dixon, Cleveland, Ohio.
Secretary: Dr. James H. Maxwell, Ann Arbor, Mich.
Treasurer: Dr. Francis E. LeJeune, New Orleans, La.
Editor, Historian, and Librarian: Dr. Edwin N. Broyles, Baltimore, Md.
Meeting: The Homestead, Hot Springs, Va., March 8-9, 1959.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. Gordon Hoople, 1100 E. Genesee St., Syracuse 10, N. Y.
President-Elect: Dr. Theo. E. Walsh, 640 So. Kingshighway, St. Louis 10, Mo.
Secretary: Dr. C. Stewart Nash, 700 Medical Arts Bldg., Rochester 7, N. Y.
Annual Meeting: The Homestead, Hot Springs, Va., March 10-11-12, 1959. (Mornings only).

Meetings of the Sections:

Eastern: Savoy Plaza, New York, N. Y., January 8-9, 1959.
Council: Savoy Plaza, New York, N. Y., January 10, 1959.
Middle: Park Plaza Hotel, St. Louis, Mo., January 12-13, 1959.
Western: The Ahwahnee Hotel, Yosemite Valley, Calif., January 17-18, 1959.
Southern: Academy of Medicine, Hotel Atlanta Biltmore, Atlanta, Ga., January 23-24, 1959.

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY.

Chairman: Dr. Victor R. Alfaro, Washington, D. C.
Vice-Chairman: Dr. Harold F. Schuknecht, Detroit, Mich.
Secretary: Dr. Walter E. Heck, San Francisco, Calif.
Representative to Scientific Exhibit: Dr. Walter H. Maloney, Cleveland, Ohio.
Section Delegate: Dr. Gordon F. Harkness, Davenport, Ia.
Alternate Delegate: Dr. Dean M. Lierle, Iowa City, Ia.
Meeting: Atlantic City, June 8-12, 1959.

AMERICAN OTOLOGICAL SOCIETY, INC.

President: Dr. Moses Lurie, Boston, Mass.
President-Elect: Dr. R. C. Martin.
Secretary: Dr. Lawrence R. Boies, University Hospitals, Minneapolis 14, Minn.
Meeting: The Homestead, Hot Springs, Va., March 13-14, 1959.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY.

President: Dr. Joseph Gilbert, 111 E. 61st St., New York, N. Y.
Vice-President: Dr. Kenneth Hinderer, 402 Medical Arts Bldg., Pittsburgh, Pa.
Secretary: Dr. Louis Joel Feit, 66 Park Ave., New York 16, N. Y.
Treasurer: Dr. Arnold L. Caron, 36 Pleasant St., Worchester, Mass.

AMERICAN RHINOLOGIC SOCIETY.

President: Dr. Kenneth H. Hinderer, 402 Medical Arts Bldg., Pittsburgh 13, Pa.
Secretary: Dr. Robert M. Hansen, 1735 No. Wheeler Ave., Portland 17, Ore.
Annual Clinical Session: Chicago, Ill., October, 1959 (definite time and place to be announced later).
Annual Meeting: Chicago, Ill., October, 1959 (definite time and place to be announced later).

AMERICAN SOCIETY OF FACIAL PLASTIC SURGERY.

President: Dr. Trent W. Smith, 327 East State St., Columbus 15, Ohio.
Vice-President: Dr. Oscar J. Becker, Chicago, Ill.
Secretary: Dr. Samuel M. Bloom, 123 East 83 St., New York 28, N. Y.
Meeting: New York, N. Y., March 18 and July 15, 1959.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY.

President: Dr. Joseph W. Hampsey, Grant Bldg., Pittsburgh 19, Pa.
Secretary-Treasurer: Dr. Daniel S. DeStio, 121 S. Highland Ave., Pittsburgh 6, Pa.
Annual Meeting:

ASSOCIACAO MEDICA DO INSTITUTO PENIDO BURNIER—CAMPINAS.

President: Dr. Antonio Augusto de Almeida.
First Secretary: Dr. Alberto Galo.
Second Secretary: Dr. Alfredo Porto.
Librarian-Treasurer: Dr. L. de Souza Queiroz.
Editors for the Archives of the Society: Dr. J. Penido Burnier, Dr. Guedes de Melo Filho and Dr. Roberto Franco do Amaral.
Meetings: Twice every month, first and third Thursdays, 8:30 P.M.

ASOCIACION DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOLOGIA DE GUATEMALA.

Presidente: Dr. Julio Quevedo, 15 Calle Oriente No. 5.
First Vice-Presidente: Dr. Héctor Cruz, 3a Avenida Sur No. 72.
Second Vice-Presidente: Dr. José Luis Escamilla, 5a Calle Poniente No. 48.
Secretario-Tesorero: Dr. Horace Polanco, 13 Calle Poniente No. 9-D.

ASOCIACION DE OTO-RINO-LARINGOLOGIA DE BARCELONA, SPAIN.

Presidente: Dr. J. Abello.
Vice-Presidente: Dr. Luis Suñe Medan.
Secretario: Dr. Jorge Perellò, 319 Provenza, Barcelona.
Vice-Secretario: Dr. A. Pinart.
Vocal: Dr. J. M. Ferrando.

BALTIMORE NOSE AND THROAT SOCIETY.

Chairman: Dr. Walter E. Loch, 1039 No. Calvert St., Baltimore, Md.
Secretary-Treasurer: Dr. Theodore A. Schwartz.

BUENOS AIRES CLUB OTOLARINGOLOGICO.

Presidente: Dr. K. Segre.
Vice-Presidente. Dr. A. P. Belou.
Secretario: Dr. S. A. Aranz.
Pro-Secretario: Dr. J. M. Tato.
Tesorero: Dr. F. Games.
Pro-Tesorero: Dr. J. A. Bello.

CANADIAN OTOLARYNGOLOGICAL SOCIETY SOCIETE CANADIENNE D'OTOLARYNGOLOGIE.

President: Dr. Robert T. Hayes, 42 Cobourg St., St. John, N. B.
Secretary: Dr. Donald M. McRae, 324 Spring Garden Rd., Halifax, N. S.
Meeting: Sheraton-Brock Hotel, Niagara Falls, Ont., Oct. 9-10, 1959.

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. William F. Hubble, Decatur, Ill.
President-Elect: Dr. Charles D. Sneller, Peoria, Ill.
Vice-President: Dr. Edgar T. Blair, Springfield, Ill.
Delegate at Large: Dr. G. Leroy Porter, Urbana, Ill.
Secretary-Treasurer: Dr. Clarence A. Fleischli, Springfield, Ill.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

President: Dr. Stanton A. Friedberg, 122 So. Michigan Ave., Chicago 3, Ill.
Vice-President: Dr. Maurice Snitman, 408 So. 5th Ave., Maywood, Ill.
Secretary-Treasurer: Dr. Fletcher Austin, 700 No. Michigan Ave., Chicago 11, Ill.
Meeting: First Monday of each month, October through May.

CHILEAN SOCIETY OF OTOLARYNGOLOGY.

President: Dr. Enrique Grünwald S.
Vice-President: Dr. Agustin Estartus.
Secretary: Dr. Marcos Chaimovich S.
Treasurer: Dr. Benjamin Kapkan K.
Director: Dr. Alberto Basterrica A.

COLORADO OTOLARYNGOLOGY SOCIETY.

President: Dr. James T. Blair, Denver, Colo.
Vice-President: Dr. James Rigg, Grand Junction, Colo.
Secretary: Dr. Will P. Pirkey, Denver, Colo.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Edward A. Newell.
Vice-President: Dr. Thomas M. McCrory.
Secretary-Treasurer: Dr. James L. Baldwin, 1627 Medical Arts Bldg., Dallas, Tex.

FEDERACION ARGENTINA, DE SOCIEDADES DE OTORRINOLARINGOLOGIA.

Secretary of the Interior: Prof. Dr. Atilio Viale del Carril.
Secretary of the Exterior: Dr. Aldo G. Remorino.
Secretary Treasury: Prof. Dr. Antonio Carrascosa.
Pro-Secretary of the Interior: Prof. Dr. Carlos P. Mercandino.
Pro-Secretary of the Exterior: Prof. Dr. Jaime A. del Sel.
Pro-Secretary of the Treasury: Dr. Jorge Zubizarreta.

FIRST CENTRAL AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY.

President: Dr. Victor M. Noubleau, San Salvador.
Secretary-Treasurer: Dr. Hector R. Silva, Calle Arce No. 84, San Salvador, El Salvador, Central America.

FLORIDA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Edson J. Andrews.
President-Elect: Dr. G. Dekle Taylor.
First Vice-President: Dr. Kenneth S. Whitmer.
Second Vice-President: Dr. William H. Anderson, Jr.
Secretary-Treasurer: Dr. Joseph W. Taylor, Jr.

FOURTH LATIN-AMERICAN CONGRESS OF OTORINOLARINGOLOGIA.

President: Dr. Dario.
Secretary:
Meeting:

FORT WORTH EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. Van D. Rathgeber.
Vice-President: Dr. William Skokan.
Secretary-Treasurer: Dr. Paul Rockwell.

GREATER MIAMI EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. William B. Steinman.
President-Elect: Dr. James H. Mendel, Jr.
Secretary-Treasurer: Dr. H. Carlton Howard.
Meeting quarterly (March , May, October and December), on the second Thursday of the month, 6:30 P.M., at Urmey Hotel, Miami.

INTERNATIONAL BRONCHOESOPHAGOLOGICAL SOCIETY.

President: Dr. Jo Ono, Tokyo, Japan.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa., U. S. A.
Meeting: Eighth International Congress of Bronchoesophagology, Mexico City, March 14-18, 1959.

KANSAS CITY SOCIETY OF OTOLARYNGOLOGY AND OPHTHALMOLOGY.

President: Dr. Clarence H. Steele.
President-Elect: Dr. Dick H. Underwood.
Secretary: Dr. James T. Robison, 4620 J. C. Nichols Parkway, Kansas City, Mo.
Meeting: Third Thursday of November, January, February and April.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Max E. Pohlman.
Secretary-Treasurer: Dr. Wendell C. Irvine.
Chairman of Ophthalmology Section: Dr. Carroll A. McCoy.
Secretary of Ophthalmology Section: Dr. Philip D. Shanedling.
Chairman of Otolaryngology Section: Dr. Robert W. Godwin.
Secretary of Otolaryngology Section: Dr. Francis O'N. Morris.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd., Los Angeles, Calif.
Time: 6:30 P.M. last Monday of each month from September to June, inclusive—Otolaryngology Section. 6:30, first Thursday of each month from September to June, inclusive—Ophthalmology Section.

LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY.

President: Dr. Fred D. Hollowell, Lamar Life Bldg., Jackson, Miss.
Secretary: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Meeting: Edgewater Gulf Hotel, Edgewater Park, Miss., May 15-16, 1959.

MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Chairman: Members serve as chairmen in alphabetical order monthly.
Secretary-Treasurer: Dr. Roland H. Myers, 1720 Exchange Bldg., Memphis, Tenn.
Assistant Secretary-Treasurer: Dr. William F. Murrah, Jr., Exchange Bldg., Memphis, Tenn.
Meeting: Second Tuesday in each month at 8:00 P.M. at Memphis Eye, Nose and Throat Hospital.

MEXICAN ASSOCIATION OF PLASTIC SURGEONS.

President: Dr. Cesar LaBoide, Mexico, D. F.
Vice-President: Dr. M. Gonzales Ulloa, Mexico, D. F.
Secretary: Dr. Juan De Dios Peza, Mexico, D. F.

MEXICAN SOCIETY OF OTOLARYNGOLOGY.

President: Dr. Rafael Giorgana.
Secretary: Dr. Carlos Valenzuela, Monterey 47, Mexico 7, D. F.

MISSISSIPPI VALLEY MEDICAL SOCIETY.

President: Dr. Arthur S. Bristow, Princeton, Mo.
Secretary-Treasurer: Dr. Harold Swanberg, Quincy, Ill.
Assistant Secretary-Treasurer: Dr. Jacob E. Reisch, Springfield, Ill.

NETHERLANDS SOCIETY OF OTO-RHINO-LARYNGOLOGY.
(Nederlandsche Keel-Neus-Oorheelkundige Vereeniging.)

President: Dr. H. Navis, Sonsbeekweg 6, Arnhem.
Secretary: Dr. W. H. Struben, J. J. Viottastraat 1, Amsterdam.
Treasurer: Mrs. F. Velleman-Pinto, Jac. Obrechtstr. 66, Amsterdam.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. J. C. Peele, Kinston Clinic, Kinston, N. C.
Vice-President: Dr. George E. Bradord, Winston-Salem, N. C.
Secretary-Treasurer: Dr. J. D. Stratton, 1012 Kings Drive, Charlotte 7, N. C.
Meeting:

NORTH OF ENGLAND OTOLARYNGOLOGICAL SOCIETY.

President: Mr. G. L. Thompson, 16 Ramshill Road, Scarborough, Yorkshire.
Vice-President: Mr. J. H. Otty, Frizley Old Hall, Frizinghall Road, Bradford, Yorkshire.
Secretary and Treasurer: Mr. R. Thomas, 27 High Petergate, York, Yorkshire.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. David D. DeWeese, 1216 S. W. Yamhill St., Portland 5, Ore.
Secretary-Treasurer: Dr. Paul B. Myers, 223 Medical Dental Bldg., Portland 5, Ore.
Meeting: Fourth Tuesday of each month from September through May, Henry Thiele Restaurant, 23rd and W. Burnside, Portland, Ore.

OTOSCLEROSIS STUDY GROUP.

President: Dr. E. P. Fowler, Jr., 180 Fort Washington Ave., New York 32, New York.
Secretary-Treasurer: Dr. Arthur L. Juers, 1018 Brown Building, Louisville 2, Ky.
Meeting: Palmer House, Chicago, Ill., October 11, 1959.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY.

President: Dr. H. Leroy Goss, 620 Cobb Bldg., Seattle 1, Washington.
Secretary-Treasurer: Dr. Homer E. Smith, 508 East South Temple, Salt Lake City, Utah.
Meeting:

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. Paul Holinger, 700 No. Michigan Blvd., Chicago, Ill.
Executive Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa., U. S. A.
Meeting: Seventh Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
Time and Place: Miami, Fla., March, 1960.

PHILADELPHIA LARYNGOLOGICAL SOCIETY.

President Dr. John J. O'Keefe.
Vice-President: Dr. Joseph P. Atkins.
Secretary: Dr. William A. Lell.
Executive Committee: Dr. Harry P. Schenck, Dr. Benjamin H. Shuster, Dr. William A. Lell, Dr. William J. Hitschler, and Dr. Chevalier L. Jackson.

PITTSBURGH OTOLOGICAL SOCIETY.

President: Dr. Bernard L. Silverblatt, 3500 Fifth Avenue, Pittsburgh, Pa.
Vice-President: Dr. Emory A. Rittenhouse, 203 Masonic Bldg., McKeesport, Pa.
Secretary-Treasurer: Dr. John T. Dickinson, Mercy Hospital, Pittsburgh 19, Pa.

PORTUGUESE OTORHINOLARYNGOLOGICAL SOCIETY.

President: Dr. Albert Luis de Mendonca.
Secretary: Dr. Antonio da Costa Quinta, Avenida, de Liberdale 65, 1° Lisbon.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Clifton E. Benson, Bremerton, Wash.
President-Elect: Dr. Carl D. F. Jensen, Seattle, Wash.
Secretary: Dr. Willard F. Goff, 1215 Fourth Ave., Seattle, Wash.

RESEARCH STUDY CLUB OF LOS ANGELES, INC.

Chairman: Dr. Orrie E. Ghrist, 210 N. Central Ave., Glendale, Calif.
Treasurer: Dr. Norman Jesberg, 500 So. Lucas Ave., Los Angeles 17, Calif.
Otolaryngology: Dr. Russell M. Decker, 65 N. Madison Ave., Pasadena 1, Calif.
Ophthalmology: Dr. Warren A. Wilson, 1930 Wilshire Blvd., Los Angeles 57, Calif.
Mid-Winter Clinical Convention annually, the last two weeks in January at Los Angeles, Calif.

SECTION OF OTOLARYNGOLOGY OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA.

Chairman: Dr. J. L. Levine.
Vice-Chairman: Dr. Russell Page.
Secretary: Dr. James J. McFarland.
Treasurer: Dr. Edward M. O'Brien.
Meetings are held the second Tuesday of September, November, January, March and May, at 6:30 P.M.
Place: Army and Navy Club, Washington, D. C.

SCOTTISH OTOLARYNGOLOGICAL SOCIETY.

President: Dr. F. T. Land, 13 Newton Place, Glasgow, C. 3.
Secretary-Treasurer: Dr. J. F. Birrell, 14 Moray Place, Edinburgh.
Assistant Secretary: Dr. H. D. Brown Kelly, 11 Sandyford Place, Glasgow, C. 3.

SOCIEDAD COLUMBIANA DE OFTALMOLOGIA Y OTORRINOLARINGOLOGIA (BOGOTA, COLUMBIA).

Presidente: Dr. Alfonso Tribin P.
Secretario: Dr. Felix E. Lozano.
Tesorero: Dr. Mario Arenas A.

SOCIEDAD CUBANA DE OTO-LARINGOLOGIA.

President: Dr. Reinaldo de Villiers.
Vice-President: Dr. Jorge de Cárdenas.
Secretary: Dr. Pablo Hernandez.

SOCIEDAD DE ESTUDIOS CLINICOS DE LA HABANA.

Presidente: Dr. Frank Canosa Lorenzo.
Vice-Presidente: Dr. Julio Sanguily.
Secretario: Dr. Juan Portuondo de Castro.
Tesorero: Dr. Luis Ortega Verdes.

SOCIEDAD DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOSCOPIA DE CORDOBA.

Presidente: Dr. Aldo Remorino.
Vice-Presidente: Dr. Luis E. Olsen.
Secretario: Dr. Eugenio Romero Diaz.
Tesorero: Dr. Juan Manuel Pradales.
Vocales: Dr. Osvaldo Suárez, Dr. Nondier Asis R., Dr. Jorge Bergallo Yofre.

SOCIEDAD DE OTO-RINO-LARINGOLOGIA, COLEGIO MEDIO DE EL SALVADOR, SAN SALVADOR, C. A.

President: Dr. Salvador Mixco Pinto.
Secretary: Dr. Daniel Alfredo Alfaro.
Treasurer: Dr. Antonio Pineda M.

SOCIEDAD ESPANOLA DE OTORRINOLARINGOLOGIA.

Presidente: Dr. D. Adolfo Hinojar Pons.
Vice-Presidente: Dr. D. Jose Perez Mateos.
Secretario General: Dr. D. Francisco Marañés.
Tesorero: Dr. D. Ernesto Alonso Ferrer.

SOCIEDAD MEXICANA DE OTORRINOLARINGOLOGIA

Monterrey 47-201
Mexico 7, D. F.

President: Dr. Rafael Giorgana.
Secretary: Dr. Carlos Valenzuela.
Treasurer: Dr. Benito Madariaga.
First Vocal: Dr. Rafael González.
Second Vocal: Dr. Juan Oberhauser.

SOCIEDAD NACIONAL DE CIRUGIA OF CUBA.

Presidente: Dr. Reinaldo de Villers.
Vice-Presidente: Dr. César Cabrera Calderin.
Secretario: Dr. José Xirau.
Tesorero: Dr. Alfredo M. Petit.
Vocal: Dr. José Gross.
Vocal: Dr. Pedro Hernández Gonzalo.

SOCIEDAD OTO-RINO-LARINGOLOGIA DE LOS HOSPITALES DE MADRID.

Presidente: Dr. Don Fernando Beltrán Castillo.
Secretario General: Dr. Don Alfonso Vassallo de Mumbert.
Tesorero: Dr. Don Rafael Garcia Tapia.

SOCIEDAD VENEZOLANA DE OTORRINOLARINGOLOGIA.

Presidente: Dr. Gabriel Briceño Romero.
Vice-Presidente: Dr. Silvestre Rincón Fuenmayor.
Secretario General: Dr. Oscar Bustamante Miranda.
Tesorero: Dr. Arturo Marrero Gómez.
Vocales: Dr. Miguel Octavio Russa, Dr. Benjamin Briceño, Dr. Oscar Gonzalez Castillo.

SOCIEDADE DE OFTALMOLOGIA E OTORRINOLARINGOLOGIA DO RIO GRANDE DO SUL.

President: Dr. Ivo Adolpho Kuhl.
Secretary: Dr. Decio Lisboa Castro.
Treasurer: Dr. Jorge Valentin.

SOCIEDAD PANAMENA DE OTORRINOLARINGOLOGIA.

Presidente: Dr. Manuel Preciado.
First Vice-Presidente: Dr. Alonso Roy.
Second Vice-Presidente: Dr. Carlos Arango Carbone.
Secretario: Dr. María Esther Villalaz.
Tesorero: Dr. Ramòn Crespo.

SOCIEDADE PORTUGUESA DE OTORRINOLARINGOLOGIA E DE BRONCO-ESOFAGOLOGIA.

Presidente: Dr. Alberto Luis De Mendonca.
Vice-Presidente: Dr. Jaime de Magalhaes.
1.° Secretario: Dr. Antonio da Costa Quinta.
2.° Secretario: Dr. Albano Coelho.
Tesoureiro: Dr. Jose Antonio de Campos Henriques.
Vogais: Dr. Teofilo Esquivel.
Dr. Antonio Cancela de Amorim.
Sede: Avenida da Liberdade, 65, 1°, Lisboa.

SOCIETY OF MILITARY OTOLARYNGOLOGISTS.

President: Lt. Col. Stanley H. Bear, USAF (MC), USAF Hospital, Maxwell (Air University), Maxwell Air Force Base, Ala.
Secretary-Treasurer: Capt. Maurice Schiff, MC, USN, U. S. Naval Hospital, Oakland, Calif.
Meeting:

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. F. R. Price, 118 Rutledge Ave., Charleston, S. C.
President-Elect: Dr. L. D. Lide, 161 W. Cheves St., Florence, S. C.
Vice-President: Dr. R. E. Livingstone, 1505 Main St., Newberry, S. C.
Secretary-Treasurer: Dr. Roderick Macdonald, 330 E. Main St., Rock Hill, S. C.
Meeting: Jointly with the N. C. Society of Eye, Ear, Nose and Throat. Next joint annual meeting of The South Carolina Society of Ophthalmology and Otolaryngology and the N. C. Eye, Ear, Nose and Throat Society at Charleston, S. C., Sept. 14, 15 and 16, 1959.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Chairman: Dr. V. Eugene Holcombe, Charleston, W. Va.
Chairman-Elect: Dr. G. Slaughter Fitz-Hugh, Charlottesville, Va.
Vice-Chairman: Dr. George M. Haik, New Orleans, La.
Secretary: Dr. Mercer G. Lynch, New Orleans, La.

VIRGINIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Benjamin Sheppard, 301 Medical Arts Building, Richmond, Va.
President-Elect: Dr. Emanuel U. Wallerstein, Professional Building, Richmond, Va.
Vice-President: Dr. Calvin T. Burton, Medical Arts Building, Roanoke, Va.
Secretary-Treasurer: Dr. Maynard P. Smith, 600 Professional Building, Richmond, Va.

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. James K. Stewart, Wheeling, W. Va.
Secretary-Treasurer: Dr. Frederick C. Reel, Charleston, W. Va.
Annual Meeting:

NOTICE TO CONTRIBUTORS

THE LARYNGOSCOPE reserves the right of exclusive publication of all articles submitted. This does not preclude their publication in Transactions of various Societies.

Manuscripts should be typewritten, double spaced, on one side of paper only and with sufficient margins to allow for corrections.

Author's name and city should appear directly under title on first page; street address at end of article.

All prints or photographs to be submitted in black and white, in good sharp contrast. Good halftones depend upon clear photographs. Line drawings for zincs to be in black and white. Colored inks or red or blue quadrille rulings will not reproduce.

References should be complete: author's surname, initials, title of article, Journal, volume, page, month, year.

Six illustrations will be furnished for each article without cost to author. Authors will please limit illustrations to six or assume the expense of additional illustrations.

Proofs will be submitted to authors for corrections. If these are not returned, articles will be published as corrected in this office.

Reprints will be furnished at the following prices:

WITHOUT COVER

	250 Copies	500 Copies	1000 Copies	2000 Copies
Four Pages	$ 19.25	$ 23.00	$ 30.75	$ 44.50
Eight Pages	33.50	42.75	58.50	83.00
Twelve Pages	47.00	60.75	86.25	131.50
Sixteen Pages	61.00	78.75	98.75	146.75
Twenty Pages	76.00	96.25	129.50	187.25
Twenty-four Pages	88.75	112.50	150.00	217.25
Twenty-eight Pages	97.50	123.25	162.25	233.50
Thirty-two Pages	115.00	139.75	180.00	267.00

WITH COVER

Four Pages	$ 37.25	$ 46.50	$ 61.50	$ 88.75
Eight Pages	51.50	66.25	89.25	127.25
Twelve Pages	65.00	84.25	117.00	175.75
Sixteen Pages	79.00	102.25	129.50	191.00
Twenty Pages	94.00	119.75	160.25	231.50
Twenty-four Pages	106.75	136.00	180.75	261.50
Twenty-eight Pages	115.50	146.75	193.00	277.75
Thirty-two Pages	133.00	163.75	210.75	311.25

Express charges will be paid by consignee.

More than 2000

RADIUM

MONEL METAL

NASOPHARYNGEAL APPLICATORS

have been delivered by the Radium Chemical Company to otolaryngologists in the United States within a period of the last fourteen years.

Results are reported satisfactory when the use of the applicator is restricted to the one purpose for which it was constructed: *the treatment of hyperplastic lymphoid tissue, which cannot be surgically removed, at the orifice of the eustachian tube or in the fossa of Rosenmueller.*

Available through purchase or annual lease at a reasonable monthly rental.

Write for complete details. No obligation.

RADIUM CHEMICAL COMPANY, INC.
161 East 42nd St., New York 17, N. Y.

SERVING THE MEDICAL PROFESSION SINCE 1913.